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LIEUTENANT-COLONEL H. WATKINS PITCHFORD, F.R.C.V.S., J.P.

COLONEL WATKINS PITCHFORD was born at Tatten Hall, Cheshire, in 1866, and is the third son of the Rev. J. W. Pitchford, Vicar of St. Jude's, London, S.E., a representative of an old Shropshire family that is mentioned in the Doomsday Book. He was educated at the St. Olive's Grammar School, London, and the Royal Veterinary College, Camden Town, graduating on May 16, 1889, and taking the Fellowship in 1894. For some years he practised in the neighbourhood of Sandhurst, but, being offered the post of Principal Veterinary Surgeon to the Colony of Natal, he sailed for Durban in June, 1906.

At that period the Colony was threatened with rinderpest, and Colonel Pitchford was at once called upon to combat the disease. The undertaking was an arduous one, as there was little or no machinery, either legal or executive, in existence beyond some dozen stock inspectors, with whom his predecessor, Mr. Wiltshire, had for many years fought single-handed an uphill fight. He at once grappled the situation and made a journey to the north of the Transvaal to meet Dr. Theiler, of Berne, who at that time was Veterinary Adviser to the Transvaal Republic. A joint scientific investigation of the disease, which had hitherto baffled any but the "stamping out method," was arranged and carried out in the Rustenburg district, whither a complete field laboratory, with all its accessories, was transported by ox-waggon, there being no railways in the country in those days.

Colonel Pitchford's and Dr. Theiler's efforts were so successful that on December 12, 1896, the former was able to report to the Natal Government: "We have discovered a process by which undoubted immunity can be conferred, and I see no reason why the progress of the disease in South Africa should not be arrested by its means." The process alluded to is the present "serum system," the best of all the methods devised. It has stood the practical test of time, and there is not a shadow of doubt, that, in spite of numerous

claimants for the honour, Colonel Pitchford and Dr. Theiler were the first discoverers of it.

To Colonel Pitchford also is due the credit of the organisation of the Natal Civil Veterinary Department on its present lines, and the Colony shortly after his arrival was divided into nine or ten districts, each under the charge of a qualified veterinary surgeon.

Shortly before the outbreak of the South African War he was asked by the Imperial authorities to undertake the formation of a veterinary corps for service in the field. The Civil Veterinary Staff responded to a man, and within a few weeks of the call for their services the Natal Veterinary Corps was enrolled, equipped, and in the field. The corps served throughout the war, and did yeoman service in helping out the sorely tried Army Veterinary Department, which from years of apathy and neglect, combined with systematic snubbing from the War Office, had been allowed to sink into an absolutely inefficient condition. The Natal Veterinary Corps is now part of the Militia Defence Force of the Colony, under the command of Major Woollatt, F.R.C.V.S., who assumed charge when Colonel Pitchford was appointed Principal Veterinary Officer on the permanent paid staff of the Natal Militia Defence Force. About the same time he was appointed Bacteriologist and Director of the Research Laboratory at Pietermaritzburg, and Analyst to the Colonial Government, giving over charge of the Natal Civil Veterinary Department to Major Woollatt. The work of the laboratory is of a most important nature, most of the preventive and curative serums, vaccines, calf lymph, &c., being prepared there; and the magnitnde of the work will be realised by the fact that 40,000 doses of vaccine for quarter evil alone were distributed in Natal during the past year. Bacteriological work proper, such as the diagnosis for tuberculosis, enteric fever, glanders, diphtheria, &c., are matters of daily routine, and this work is daily increasing.

Many valuable reports have been lately issued from the laboratory under Colonel Pitchford's direction, subjects such as "Horse Sickness," "The Characters of the Mark VI. Service Bullet," "The Bacteriology of the Plague Bacillus," "A Review of the Efficacy of Modern Disinfectants," "A Report on the Use of Copper Salts in the Treatment of Water Supplies," and "A Comparison of the Snake Venoms of Natal." The mention of these titles will serve to show the wide field of usefulness covered by this institution.

In addition to his other appointments, Colonel Pitchford is a Justice of the Peace, and the Analyst to the Corporation of Pietermaritzburg.

Editorial.

PARLIAMENTARY REPRESENTATION.

THE meeting of delegates appointed to consider ways and means for obtaining parliamentary representation has not been quite as successful in its results as had been hoped for by many members of the profession ; at the same time, it has made a distinct step in the forward direction, in that it has appointed a committee with powers to take any steps considered advisable to obtain direct or indirect representation in the House of Commons. It now remains for the various Societies to send delegates with distinctly definite instructions as to the manner in which they shall support any scheme which will help forward the return of a veterinary surgeon as Member of Parliament.

The Central Veterinary Medical Society of London have considered the subject of sufficient importance to authorise their President to convene a special meeting to discuss the subject. There is no doubt that it will be thoroughly well ventilated and thrashed out before the delegates are appointed, as this Society is a large and powerful one, and it is very rare to find any subject upon which all its members see alike.

It is to be hoped now that the matter has been started every Veterinary Society in the kingdom will devote special attention to this question, and that the result will be that some practicable and workable scheme will be formulated whereby, on the first possible opportunity, a gentleman possessing the membership of the Royal College of Veterinary Surgeons will be able, in addition, to add M.P. after his name.

In fact, it is to be hoped that the matter will not rest there, and that the result of the next election will find not only one, but several successful candidates.

A few years ago the idea of a medical man in Parliament would have been laughed at, but now he is looked upon quite as a necessity. The present election has shown, by its results in the Labour Party, what determination combined with organisation can do, and there is not the slightest reason why, if we act up to our motto of *vis unita fortior*, we should not achieve an equally satisfactory result.

Original Article.

GLANDERS IN HORSE AND MAN.

BY WILLIAM HUNTING, F.R.C.V.S.

(A Paper read before the Epidemiological Society, Friday, January 19, 1906.)

THERE are three diseases—anthrax, rabies, and glanders—which annually destroy a number of animals and human beings, but which never attack man unless by infection from an animal. If, then, these diseases can be stamped out among animals they cease to injure man.

Anthrax, rabies, and glanders have for many years been scheduled under the Diseases of Animals Act, with the result that rabies has been stamped out and hydrophobia has ceased in Great Britain. Anthrax, according to the Government Returns, has slightly increased. Glanders remains much about the same as when the existing regulations were framed in 1894.

Had these three diseases not been communicable to man it is doubtful whether any of them would have been scheduled. Diseases of animals are disregarded by Government Departments, until some public agitation and some collective pressure is brought to bear, insisting upon their control. The interests of stock-owners are well looked after by agricultural societies, and Government moves accordingly. Diseases of dogs existed for years without control, and it was only when the medical profession took up the question of rabies, that any proper regulations were enforced. The horse-owner is represented by no collective bodies able to voice his wants and grievances. If he is galvanised into temporary activity for the protection of his property, each man states his own claim and insists upon his own panacea. The result is, that Government replies: "There is such a diversity of opinion as to what is necessary, that we cannot yet frame any further regulations." Eventually the horse-owner has come to recognise the expediency of stamping out glanders—the veterinary profession has given unanimous advice as to how this may be done, and the most important local authorities in the kingdom have urged the Government to adopt the necesary measures. The Board of Agriculture has ceased to invent excuses for doing nothing, and is prepared to issue a new Order; but the Treasury declines to find the funds.

So long as regulations are not clamoured for by those most interested, and so long as property only is affected, the Treasury hides easily behind a *non possumus*. But if it can be shown that human life is affected, an appeal for protection cannot be refused, provided there is some reasonable proportion between the cost and the number of lives to be saved.

This, shortly, is the case which I wish to represent to the Epidemiological Society; and I propose to adduce evidence proving the frightful loss of horses, the loss of men much greater than is acknowledged, and the ease and economy with which glanders might be stamped out.

Glanders in the Horse.

Prevalence.—Although glanders has been scheduled since 1869, we have no very reliable statistics until about 1881. From that year to the end of 1905 the Returns show a loss of 44,943 horses. This is an annual average of nearly 2,000; and it will be evident that no progress is indicated in the control of the disease, when I say that the number attacked in 1881 was 2,110, and last year 2,062. We have had worse years than these—for instance, in 1892, 3,001 horses were attacked, and in 1904, 2,628.

The disease exists chiefly in the large towns London and Glasgow being the two great centres. London shows 75 per cent. of all the cases reported; and the only other counties that are never free are those immediately surrounding the Metropolis. If we group London with Middlesex, Essex, Kent, Surrey and Hertford, we have a district in which 90 per cent. of all reported glanders is found. This distribution of the disease is important, because it strongly suggests that regulations would be more certainly effective than if glanders prevailed generally.

Modes of Infection. — The causal organism may enter the body through the skin, through the air passages, and by the digestive tract. Direct experiment has shown that inoculation and ingestion are modes of infection. I am not prepared to deny that inhalation may be another mode, but I know no evidence to support it. Clinical observation suggests that ingestion is by far the most common way in which glanders enters the system of the horse. No infection spreads, except from a living diseased horse; and if we were permitted to find these living centres, to isolate them and destroy them, the progress of the disease would be stopped.

Period of Incubation.—This is difficult to arrive at, because lesions may form in the lungs without the accompaniment of any clinical signs. If the period be reckoned from the time of infection to the time at which definite lung lesions are formed, we might put it at from two weeks to two months. If we mean by "period of incubation" the time which elapses between infection and the appearance of clinical symptoms, the duration is quite indefinite. Probably three months is about the average time; but I have known many cases in which a year or more has passed before clinical symptoms have been recognised.

Duration of the Disease.—This also is indefinite in horses. Acute cases with pneumonia or pleurisy are fatal in a few days. Cases without much constitutional disturbance may last for weeks, and then all symptoms disappear. I have known many horses apparently recover, and one in which, after two months' continuance of symptoms, an apparent recovery resulted; but, after eight years' work in an omnibus, there was a good reaction to mallein, and clear *post-mortem* lesions of the disease. with no outward sign of infection.

Symptoms in the Horse.—The typical signs of glanders are enlargement of a submaxillary gland, nasal discharge—often unilateral—and ulceration of the nasal membrane. With these we may have swelling of one or more legs, and the appearance of small round tumours on and under the skin, the so-called farcy buds. These buds may be solitary or in groups. In the latter case they are usually connected

by enlarged lymphatics, the so-called farcy cords. The old distinction between glanders and farcy is not worth while retaining, because it is seldom we find cases in which a mixture of symptoms does not exist; and besides, both sets of symptoms are due to the same infection.

The division of cases into acute and chronic is also arbitrary. Fever and constitutional disturbance exist to some extent in nearly every case. When they abate a chronic stage may exist, but existing regulations prohibit horses so affected remaining alive.

It is not common in horses to find symptoms result from lesions in joints, and it is very rare to see any signs of trouble in the muscles.

Post-mortem Lesions.—The only definite constant lesion is the nodule found in the lungs. With the possible exception of those few cases of direct inoculation through the skin which give rise to local farcy sores, we find pulmonary nodules in every horse presenting clinical signs of glanders. In a few cases nodules are found in the liver and spleen, very rarely in the kidney.

Ulceration of the nasal membrane is usual, of the trachea frequent, and of the larynx less common. Enlarged glands are not often found in the thorax, and very seldom in the abdomen. The bronchial and tracheal glands usually contain some small nodules, with caseous centres. It is remarkable that in glanders the bronchial glands are seldom enlarged, whereas in tuberculosis of the lungs they invariably are.

Of 1,000 *post-mortem* examinations made by veterinary inspectors of the London County Council, 462 presented nodules in the lungs as the only internal lesions. The nasal membrane was ulcerated in 378, the larynx in 179, and the trachea in 201.

Method of Spread.—Experience has shown that if a visibly glandered horse be kept in a stable with other horses the disease will spread. Given a glandered horse with an infective discharge, the disease is spread through nosebags, pails, mangers, or other objects which convey the infection to healthy horses. That the spread is by ingestion has many facts in its favour; to the theory that aërial infection may take place the clinical facts give no support. My experience leads me to believe that an infected horse does not infect others until some clinical signs of disease are apparent. There is just one kind of case that must be taken as an exception to this, viz., when ulceration of the trachea exists, with no other clinical sign or *post-mortem* lesion, except nodules in the lung. Such cases are rare, but are sometimes found at an autopsy, and must be recognised as possible. The mere existence of glanders nodules in the lung does not constitute an infective condition; but as such an animal may, at very short notice, develop lesions, accompanied by an escape of bacilli, he is always dangerous.

Mallein.—The discovery of mallein in 1890 by a Russian veterinary surgeon has altered and simplified the whole process of preventing and suppressing glanders. Before it was known we had to wait for the appearance of clinical symptoms before we could detect disease. As these symptoms were often slight or dubious, infection of other animals frequently took place before a diagnosis was made.

With mallein we can with certainty detect all cases in a stable before they have reached the infective stage, and we can verify a doubtful diagnosis.

As you know, mallein is an extract of the *Bacillus mallei*—an analogous material to the tuberculin derived from *Bacillus tuberculosis* The dose for a horse is about twenty drops, injected under the skin, usually on the side of the neck. If the horse be free from glanders no reaction takes place. If it be infected, the injection is followed by a rise in the animal's temperature, and by a swelling at the place of injection. This double reaction renders the diagnostic aid of mallein much more reliable than the mere temperature rise given by tuberculin. It is worthy of notice that the reaction of mallein in glandered horses bears no proportion to the extent or duration of the disease. It discloses the existence of infection, and thereby enables us to detect and isolate every dangerous horse in a stud. This action permits the removal of every infected horse, and the continuance at work of all the healthy.

As the spread of glanders from stable to stable is almost always the result of bringing into the healthy stable a horse suffering from latent glanders—a horse apparently healthy—we have in mallein an agent which, with other compulsory regulations, renders it possible to check the spread of disease at once.

The regulations at present in force are quite inadequate. They provide compulsory notification by the owner or occupier, diagnosis by a veterinary inspector, and slaughter, with compensation, of all diseased horses. They do not provide for any action over the in-contact horses. A stable of a hundred horses may show a case of glanders every week for months, a condition which renders it certain that many are infected, though not visibly diseased. At any time this stud may be sold, and carry unsuspected disease into a dozen other stables. This is the chief alteration required in the regulations—power to deal with in-contacts, and to detect them by testing every infected stud with mallein.

It is argued by some horse-owners that the compulsory slaughter of a horse which exhibits no disease, which may never show any, and which is of great value to him, is unjust unless fair compensation is given. I agree with this, and so do the majority of local authorities. I think I may say that the Board of Agriculture also acknowledge its justice. And here is the obstacle to progress. Funds are required, and the Treasury will not supply them.

I venture to think that the loss of 2,000 horses a year is worth the expenditure of a few thousands; and that the additional arguments of danger to the efficiency of our Army, and an annual loss of human life, make the case unanswerable.

Glanders in Man.

And this brings me to glanders in man—an attempt to arrive at the loss so incurred. Three times the question has been asked in Parliament: "Whether the danger from glanders to human beings does not require more attention and further legislation for its prevention." Each time the answer has been the same: "That glanders is very rare in man, the Registrar-General's returns only showing about two deaths per annum, rising in a recent year to four, and once to six."

I am prepared to admit that if only two human beings per annum lost their lives from glanders, a very poor case would exist for any

costly and harassing regulations. But enquiry leads me to think that the official returns do not nearly represent the deaths due to glanders. I have collected a number of reported cases from the medical journals and the lay Press, and find that even when these occur in public institutions, diagnosis is very uncertain. No cases are reported by private practitioners, and yet they most surely see some. The clinical diagnosis in public institutions has included rheumatic fever, typhoid, small-pox, syphilis, blood poisoning, and influenza. The correct diagnosis has only been arrived at on the day before death, or by the aid of bacteriology the day after. It is suggested to me that in the majority of cases of glanders in man, the disease is not diagnosable without the bacteriologist, and, therefore, that most of the fatal cases occurring in private practice are registered under some other name.

The following are some notes of cases :—

I. Inquest on George Chinn, at Lambeth, December 12, 1905. The man was a farrier, and worked in a cab-yard. Returned home on November 21, and said to his wife, "I've got glanders." She saw blood and matter from his nose. Dr. Councell was called in on the 24th. He saw no blood or matter. The patient complained of general malaise, but there were no localised symptoms. The witness noticed hæmorrhagic spots on both legs upward as far as the knees, and a foul ulcer on the right forearm. In the absence of other signs, witness came to the conclusion that he was suffering from septic absorption in consequence of this foul ulcer.

On November 29 the patient was admitted to St. Thomas's Hospital, and was seen by Dr. Dean on December 3, who said his temperature was 103°, and his pulse very rapid. On the hard palate and on the tongue were greenish-yellow patches. The legs were covered by large hæmorrhagic patches. The patient was not admitted for glanders, and the disease was not at that time considered. Owing to the hæmorrhagic spots which appeared from day to day, the physician in charge came to the conclusion that it was a case of glanders. The man died on the 8th.

A *post mortem* made on the 11th disclosed no evidence of anything in the nature of glanders in the lungs. They did not find the specific bacillus. They found two ulcerated patches in the larynx, but these might be found in any septicæmic process. He was of opinion that death was due to an acute septicæmic process, in consequence probably of the infection of glanders. The jury returned an open verdict.

II. Inquest on Albert Tharby, at the London Hospital, early in September, 1905. The man was a carman, and felt unwell on August 5. Treated by Dr. Sergeant at his surgery daily till the 10th. On the 13th, doctor visited him at his house. Found large red spot on each knee, with pains in the joints. Said the case was one of septic pneumonia, and patient had better go to hospital, which he did next day. Died on August 30. Verdict: Death from glanders. This man only drove one horse; did not clean or feed any others. The horse he drove was tested with mallein, and gave no reaction.

III. These two cases were reported in the *Lancet* by Dr. Goodall. A man, aged 55, a printer, was admitted into the Eastern Fever Hospital on October 28, 1903. He was seized with shivering and pain in the limbs on the 9th. On the 11th he was worse, and was medically attended at home till the 20th. On that date he went as out-

patient to a general hospital, whence he was sent as a case of typhoid fever to the Eastern Hospital.

On admission he was semi-comatose. On the skin were a number of small superficial pustules, most abundant on the chest, right leg, and right forearm. There were a few on the left cheek, the back, left thigh, and left upper arm. Besides these pustules there were several swellings of varying size in different places—one on the right frontal eminence, two on the right forearm, two on the left forearm, one on the right leg, and one on the left. The largest swellings fluctuated, the smaller did not. Some appeared to be in the subcutaneous tissue, others were distinctly in muscles. There was no discharge from the nose. On the morning of the 30th the contents of a swelling on the right leg were drawn off and microscopically examined. A bacillus resembling that of glanders was at once detected. The patient died on the 31st.

The second case was that of a stableman, who was taken ill with "pains all over," ten days before admission. He was sent to a general hospital as a case of blood poisoning, and then to the Eastern Hospital as one of typhoid fever. In one of the muscles of the left forearm was a nodular swelling. The left knee-joint was much swollen, very painful, and distended with fluid. The patient said that the lump in the arm had been present since February 18, and the knee-joint had been affected since the 16th.

Next day an examination of fluid from the knee and arm was made, but no organisms were found. Cultivations in agar were made, and, on the second day, what appeared to be the *Baccillus mallei* was detected. A day or two later Drs. Lewis and Wood obtained the bacillus from the fluid of the knee-joint. The patient died delirious on March 11.

IV. An inquest was held at Westminster, on August 15, on Albert Allen, a horse-keeper in an omnibus yard. Dr. Cope saw the patient first on June 12, when he showed symptoms of an ordinary cold, with aching of the limbs. He was treated for a rheumatic attack. On the 19th there was a nodule on the leg, and some inflammation of the limb. On July 12 other nodules appeared on the right arm, and on the 13th the man was taken to Westminster Hospital.

Mr. Frank Mott saw him on the 14th; and, as there was a suspicion of glanders, some pus was examined, and the *Bacillus mallei* discovered. The man died August 11. An autopsy revealed eleven abscesses scattered over the arms and legs. There was pus in the knee-joint. Nothing was found in the nose, throat, larynx, or trachea; but in the lungs some small nodules, about the size of a pea, were discovered.

V. In a note published by Drs. Bullock and Twort, of the London Hospital, the first sentence is: "Glanders in man is generally believed to be uncommon, although, no doubt, the belief is partially due to the protean characters which the disease manifests clinically, and which render it difficult of diagnosis." They then give some details of six cases, which had come under their notice at the hospital, and of a seventh case which was of a doubtful character. Four of these cases were chronic, and characterised by recurring abscesses which lasted for months.

VI. Inquest on Charles Nicholls, a horse-keeper, at Westminster,

May 19, 1905. On May 1 the man complained of illness, and saw a doctor, who said he had pleurisy. Two or three days later he was taken to St. George's Hospital, where he died on the 16th. Dr. Hunt said he diagnosed the case as rheumatic fever. Dr. Ethrington, house physician, said the deceased, on admission, seemed to be suffering from rheumatic fever. The day before death it was thought to be glanders, and a *post-mortem* examination verified the diagnosis.

VII. From the *Lancet* of April last I take the following from a report by Mr. M. J. Cromie, House Surgeon, Westminster Hospital. A horse-keeper, aged 25, was admitted on November 12, 1904. A consultation between Dr. Hebb and Mr. Spencer led to a surmise that the patient was suffering from glanders. On the following day Dr. Hebb found the *Bacillus mallei*. The patient died thirty-three hours after admission. A full consideration of the case exposes possibilities of infection, and of such cases being overlooked is Mr. Cromie's comment, and the history more than bears this out. On November 3 he had pain and swelling of the left side of face, and was advised by a dentist to bathe his face in hot water, as an abscess was forming. He next saw two other medical men, who told him that he had cellulitis and erysipelas. On the 9th small hard lumps began to appear in the neighbourhood of the joints, with pain in the joints, especially the knees. On this day the patient went to a hospital, and, on his out patient paper was entered cellulitis of the face. An incision was advised; but, as he could not be admitted and felt very ill, he declined it. On the 10th the nodular swellings became red and inflamed, and pustules appeared on the swollen face, the lips, and the buccal aspects of the lips and cheek. The lips became black, nasal discharge commenced, and dyspnœa supervened, He was therefore taken to another hospital, when he was given a paper saying he was suffering from cellulitis and cephalitis (*sic*). Finally he was taken in an ambulance to the Westminster Hospital.

The *post-mortem* examination showed abscesses in the muscles all over the body. The mucosa of the nose and sinuses was much swollen, but there was no ulceration of the nasal membrane. The lungs were engorged, and the seat of numerous abscesses.

VIII. An inquest was held at Ipswich, on September 26, on Edward Rogers, an ostler. On September 5 he received a wound on his right hand in balling some horses. On the 12th he went to work at another stable, but in two days the arm became swollen, and he attended as an out-patient at the hospital. Dr. Rygate then attended and treated the case as one of blood poisoning, till the 23rd, when a consultation was held with the Medical Officer of Health, and the man taken to the Fever Hospital. He died delirious on Sunday, the 25th.

No *post-mortem* examination was made, which is unfortunate, as the case was one of direct inoculation, and some description of the lymphatic glands on the injured limb would have been helpful.

IX. On May 14, 1903, an inquest was held at the Town Hall, Chelsea, on Charles Ford, an organ-grinder. The man kept his organ in a mews, but lived in a street some distance off. A case of glanders existed in a stable opposite that in which the organ was kept—about a month previous to the man being taken ill—and the mews had frequently contained glandered horses. The widow said: "It was about

a month since he first complained of pains in his head and all over his body." Dr. Cope was called in, and treated the case as one of influenza. He had no cuts or sores about him, as far as she knew. He was taken to the infirmary on April 28. Dr. Coulson, Assistant-Medical Superintendent at St. George's Workhouse Infirmary, said the man was admitted for debility after influenza. He had a high temperature, but no local symptoms, until three days before death. Then a small sore developed over the left eye. The next day there was suppuration from the nose. The *post-mortem* examination disclosed several small nodules on the skin of the face and nose. The nasal membrane was intact, as was that of the larynx and trachea. Thick, tenacious pus was found under the dura mater. A deep-seated swelling on the left forearm, on being incised, was found to be an intramuscular abscess.

X. In the *Lancet*, February, 1901, Drs. Faucet and Pakes describe a case of chronic glanders seen at Guy's Hospital. I need not quote more than one sentence. There seems to be little doubt that, had it not been for the bacteriological examination, the case would not have been certainly diagnosed as one of glanders.

XI. An inquest was held at Hammersmith, in November, 1893, on William Wallace, a horsekeeper. The widow stated that towards the end of September the deceased told her he had been bitten by a horse, and about a week later he complained of severe headache. Following this came pain in the arms and legs, and on October 7 he went to a dispensary. The pains increased, and on October 14 he went to the West London Hospital, when he was told that he was suffering from rheumatic fever. He was admitted into the hospital on the 18th, and died on the 24th. Dr. Wilks, the house physician, said the case was of a very doubtful character, and it was not till the 23rd that a diagnosis of glanders was made.

In this case Dr. C. Creighton made a full *post-mortem* examination, and wrote a pathological report, which I quote in full.

The Eruptions on the Skin.—Numerous nodules, flat or depressed, with a small white point in the centre of many of them; single, or confluent in groups, of somewhat firm or even hard consistence, varying in size from a pin's head to the diameter of a third of an inch. The largest were on the forehead and neck, the next largest on the shoulders and upper arms, and on the front of the chest; a few occurred on the forearm and hands, and a few on the thighs. They were thickest and of largest size on the right side in all the principal seats of their occurrence. Two, of the largest size, on the forehead, had sloughed, and had a deep red or purple appearance; two or three on the side of the neck were partly disintegrated; elsewhere no destructive process was observed.

There was no swelling of the eyelids. or other trace of erysipelas.

Nodules on the Mucous Membranes.—The cartilaginous septum of the nose was removed by dissecting beneath the upper lip. On the mucous membrane of one side of it was an ulcer about two or three lines in diameter, deep red, with several minute white granules in its floor. On the other side was a flat, whitish nodule, about a line in diameter. A similar nodule was found on the under-surface of the upper lip, one of the same kind on the under-surface of the epiglottis three of the same kind in the interior of the larynx, two in the uppe

part of the trachea, with minute white points around them, and several smaller white nodules in the lower part of the trachea and in the right and left bronchii. The mucous membrane from the throat to the lungs was dusky red.

The Lungs.—The disease was confined to the upper and middle lobes of the right lung, and to the upper lobe of the left. Close to the surface could be felt numerous isolated masses, varying in size at their peripheral end or base, from a quarter to three-quarters of an inch in the longest direction. On section, they were found to be of somewhat firm consistence, greyish-white, with a cheesy broad base on the pleura and a convex margin towards the interior of the organ, resembling in all respects infarcts or formation within the vascular territory of terminal branches of the pulmonary artery. In one of those incised, the outer zone was of coagulated blood, only a small superficial area, the middle being greyish-white. They were most numerous in the right lung, being confined almost to the apex of the left; the lower lobe of the right lung was firmly adherent to the diaphragm, and was collapsed; the lower lobe of the left lung was œdematous and conjested.

Other Organs.—The right ventricle of the heart was empty. A partially decolourised clot projected from the vena cava inferior into the right auricle. The spleen was of normal colour externally, not diffluent within, of average size; the left kidney was somewhat large, its substance somewhat cloudy; the liver was normal; there was no congestion in any part of the small intestine, nor in the cæcum.

Lymphatic Glands.—The bronchial glands were normal, also the mesenteric; the glands of the right axilla were displayed by reflecting the pectoralis major muscle, and were seen hanging in a cluster of small red bodies, unaffected by the disease. The chain of posterior cervical glands were also displayed by an incision along the posterior border of the sterno-mastoid muscle, and found to be small red bodies, the same as in the axilla. The absorbent glands under the chin were also normal.

Muscles and Joints.—An incision three inches above and three inches below the bend of the right elbow, corresponding to a swollen region of the limb, revealed a remarkable infiltration of the group of pronating and flexing muscles of the forearm attached to the inner condyle; the infiltration was about three inches long and an inch and a half deep, of yellowish-white colour, somewhat firm consistence, caseous, nowhere purulent; it occurred between the several muscles, and also mottled their substance. When the capsule of the elbow-joint was punctured at the bottom of the mass, greenish-yellow pus welled out, somewhat thin.

A corresponding incision of the left elbow revealed œdema of the tissues under the skin, but no infiltration of the muscles, which looked normal in colour and consistence. When the joint was opened a thin greenish-yellow pus flowed out, as on the right side.

The Hands.—There was no trace of wound or other injury on the fingers of either hand, or on the hands generally.

The numerous nodules on the skin are the characteristic farcy-buds of a case of glanders in man, progressing somewhat slowly. They were most abundant on the right side, and of largest average size on the neck and forehead, where two of the largest had sloughed, and one or two others were partially sloughed.

The ulcer on one side of the cartilaginous septum of the nostrils, with small white granules in its floor, is distinctive of glanders in man. The yellowish-white flat nodules in the following situations, namely, the other side of the nasal septum, the under-surface of the upper lip, the interior of the larynx and the windpipe, are distinctive in their appearance and in their situation in a series.

The formations in the lungs would not be distinctive by themselves of glanders in man, inasmuch as the same peripheral masses, more or less wedge-shaped, with the broad end outwards, are found in some forms of tubercle, in which also the colour and consistence may be the same as in this case. But they are precisely of the kind usually found in the lungs in cases of human glanders, and are undistinguishable in appearance from the pulmonary nodules of glanders in the horse.

The remarkable infiltration of the pronating and flexing group of muscles of the right forearm is in a situation which M. Brouardel specifies as one of the most usual situations for such infiltration in human glanders. The substance was neither purulent nor phlegmonous, but of the same yellowish-white, somewhat firm, consistence as in the lungs. The suppuration within both elbow-joints is the same as in pyæmia.

The seat of infiltration at the right elbow was not connected by continuity with any observed disease in the lower part of the forearm, nor was there any trace of injury on the fingers of the right or left hand.

The complete immunity of the lymphatic or absorbent glands in all the affected regions is noteworthy. It points to the virus having circulated with the blood, and not in the lymphatics.

The appearances were, on the whole, those of a somewhat slow infective process, which might have lasted a month or more—neither acute or chronic, but something between.

The anatomical examination does not enable me to offer an opinion as to the way of entrance of the virus ; but I am of opinion, as indicated above, that it passed into the blood without any intervention of the lymphatic or absorbent vessels and glands. The preponderance of disease was undoubtedly on the right side of the body, both externally and internally, but the fact has no very clear significance.

C. Creighton, M.D.

Mode of Infection in Man.—It is generally believed that man is always infected by direct inoculation through the skin. The cases I have laid before you give no countenance to that theory. In only one case—that at Ipswich—was direct inoculation proved, and in that local symptoms were plentiful on the injured limb.

I should suppose that inoculation would always cause some local sign of disease before general infection appeared, and that even in the most acute case the seat of inoculation would show some abnormal changes. In many of the cases reported, a legend of a bite or a cut finger was referred to by some of the witnesses, but in none was a wound or scar found by the medical attendant, nor was any special infection of the supposed injured limb detected. The *post-mortem* examinations might be expected to show some changes in lymphatics or glands near the seat of inoculation, but this was not so.

The ways of horse-keepers are not aseptic. I have frequently seen men handle the nostrils of glandered horses, and merely wipe the pus and mucus off their hands by a little manual friction on the seat of their trousers. The wonder to me has always been how so many escaped. The facts I have mentioned force me to believe that ingestion is more often the mode of infection in man than inoculation. Meals are eaten in the cab and omnibus stables with unwashed hands, and the nasal discharges from glandered horses might be easily transferred to the men's food.

I have notes of many more cases, but I think those I have read will suffice to show that glanders in man is not easily diagnosed. In no case have I found a diagnosis made by a private practitioner. In very few cases is the disease recognised in public institutions until the day before death. In hardly any case is the diagnosis certain without bacteriological assistance. I need hardly say that I am not reflecting upon the skill of medical practitioners. My view is that in the majority of cases there is no diagnostic symptom of glanders until just before death of the patient. In the last few hours there is apparently a rapid development, which causes the appearaace of rashes of small nodules upon the skin, mentioned generally to the medical man who makes the *post mortem*.

If this difficulty of diagnosis appears in nearly every reported case, is it not logical to assume that scores of unreported cases remain undiagnosed, and that death is credited in the Registrar-General's returns to other diseases. Instead of two per annum, the deaths from glanders may be a hundred ; they are certainly many. All these lives might be saved by stamping out glanders among horses ; they can be saved in no other way. I ask the medical profession to join the veterinary in pressing upon Government the absolute necessity of altering the existing regulations under the Diseases of Animals Act, so that the only possible protection against a horrible disease may be afforded to men.

In less than twenty-five years the country has lost 50,000 horses from glanders, of a money value probably exceeding £1,000,000 sterling.

In our last war great inefficiency and loss resulted from the presence of glanders. It will be so again if nothing is done.

No one knows how many human beings have lost their lives from this disease, but we do know that they are more than was ever suspected, and that they will only cease when glanders has been stamped out among horses.

Clinical Articles.

A CASE OF POISONING BY CREOLIN.

BY T. SLIPPER, F.R.C.V.S., SUTTON COLDFIELD.

THE subject, a grey hunter gelding, received by mistake an eight-ounce bottle of creolin mixed with a pint of water. When I saw the patient (about twelve hours afterwards) the symptoms presented were those of dulness and general restlessness, as if slightly griped. The pulse was quick and the temperature two or three degrees above normal. The mouth was clammy, and the visible mucous membranes pale and dirty-looking. The horse would eat a little and drink linseed tea. The colour of the urine was the most distinguishing feature; it became very dark, eventually almost black, and with a distinct tarry odour. For three days, although dull, the horse did not seem hopelessly ill, but after that time there was great dulness, entire loss of appetite, great prostration, cold extremities, tucked-up flanks, and hurried respiration. Finally the symptoms were those which one gets in acute enteritis, and the horse died in violent abdominal pain, notwithstanding all our attempts to save him.

CALCULUS OF THE INTESTINE IN A HORSE; LAPAROTOMY.

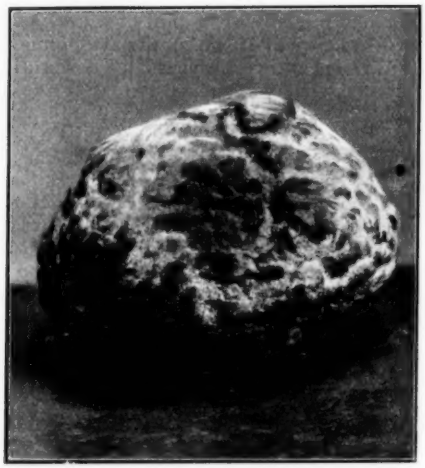
BY GEORGE C. LOWE, M.R.C.V.S., BERMONDSEY, AND FREDERICK HOBDAY, F.R.C.V.S., KENSINGTON.

THE patient was a light van mare, 19 years old, formerly in a tramway stud, and it was known that for a period of two years and a half preceding this attack she had been entirely free from colic. When first seen on December 20 last the mare had just finished a day's work, and she showed only slight uneasiness and symptoms of dull pain. The rectum was empty, and no passage of fæces took place during the night.

Next morning a large, mulberry-like calculus in the pelvic flexure of the colon could be distinctly felt through the wall of the rectum.

As the calculus could not be moved either by manipulation or by extensive irrigation of the intestine, it was decided to operate before the animal's strength had given way. Forty-eight hours after the onset the mare was cast and chloroformed, being shaved over a large area on the left flank. Before opening the side an attempt was made to reach the calculus through an incision in the vagina. This, however, failed, as although it could just be touched, it was too far forward for the hand to obtain a grasp upon it. Laparotomy was now performed, with strict attention to antiseptic details, high up in the flank, and the calculus was again reached without any trouble. It was found to be tightly wedged in the pelvic flexure of the colon, and it was only after persevering efforts, lasting over half an hour, that it was eventually moved back again into the larger lumen of colon, where it must previously have been existing for some considerable time. In the attempt it was found that it was impossible to get the portion of gut which contained the stone into the wound in order to incise it.

The mare never got up again. After the operation she remained quiet for some hours, and then began to struggle as if in a state of delirium, and on grounds of humanity was painlessly put away. At the *post-mortem* examination the calculus was found lying loose in a mass of ingesta at the end of the fourth portion of the double colon,



Photograph of the calculus.

and there was plenty of room for the passage of the contents of the bowel. Slight signs of bruising were noticeable where the massage had been applied and where the calculus had previously been jammed in the pelvic flexure. The calculus itself weighed 6 lb. 6 oz.

That occasionally a successful or partially successful result may be obtained has been shown by the result of Case 5 on page 268 of the VETERINARY JOURNAL for May, 1905, in which the patient lived for fifteen days after the calculus had been moved along the bowel from the place in which it had originally been fixed, dying ultimately from septic pneumonia, and having no peritonitis in the abdomen.

[This case forms number six of a series of attempts in this direction, five of which have already been published in the VETERINARY JOURNAL for March and May, 1905.—ED.]

DISPLACEMENT OF THE FIFTH UPPER MOLAR TOOTH INTO THE NASAL CAVITY.

BY J. J. O'CONNOR, M.R.C.V.S., PROFESSOR OF SURGERY, ROYAL VETERINARY COLLEGE OF IRELAND.

A GOOD-LOOKING grey carriage horse was sent into the College Clinique with a profuse, very offensive, purulent discharge from the right nostril and a prominent swelling on the lower third of the face;

respiration was performed noisily. The condition had been diagnosed as a probably incurable malignant disease of the nose, and all the symptoms tended to confirm this diagnosis. On inserting the finger into the right nostril its tip just came in contact with a rough, hard obstruction, which seemed to be an encroachment of the facial swelling on the nasal passage. There was no trace of foodstuff in the discharge, and the horse fed without any apparent difficulty.

Having decided to operate under chloroform, I opened the right nasal cavity at the inside of the false nostril. Inserting my finger through the opening thus made, I found that the obstruction already alluded to was movable, and on disturbing it some foul-smelling food material came into view. I then recognised that the obstruction was a molar tooth, and, using considerable force, I dislodged it by means of a retractor. The passage behind the tooth was packed with masticated food, which I removed with a surgical spoon. On examination of the mouth I found that the fifth upper molar of the right side was absent.

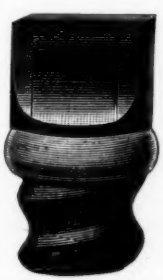
The nasal cavity was washed daily afterwards with antiseptic lotion, and the distressing symptoms greatly abated.

After about a week, seeing that food was still coming through the nostril, I trephined the frontal and superior maxillary sinuses of the affected side, and found them full of food-pulp. Having cleared them of this and irrigated them with suitable lotion, I plugged the vacant alveolus with gutta-percha. The trephine openings were kept patent for the purpose of irrigation and drainage until all traces of discharge and smell had disappeared. In about a fortnight the animal made a good recovery.

OBSTRUCTION OF THE BOWEL BY A BOTTLE-STOPPER IN A DOG: LAPAROTOMY.

BY ARCHIBALD BAIRD, M.R.C.V.S., EDINBURGH.

THE patient, a fox terrier puppy, was brought into hospital on December 12 with a history of colic, accompanied by dulness, capricious appetite and vomiting. There was a half-hearted story that the



The bottle (screw vulcanite) stopper.

dog had been seen to be playing with a beer bottle screw vulcanite stopper, and this could not now be found. For two days we treated

the patient with stomach sedatives to allay the vomiting, but on the third day digital examination of the exterior of the abdomen distinctly revealed the presence of a foreign body in the intestine. Laparotomy was decided upon, and this was performed at once under chloroform and with strict attention to antiseptic details. There was no trouble whatever about finding the piece of gut which contained the foreign body, and upon incising this in the longitudinal direction the beer bottle stopper was readily removed. The bowel was sutured with fine catgut, Lembert's pattern of suture being applied, and the abdominal wall was closed with a double row of interrupted silk sutures. The patient was fed carefully during the next week, and made a most excellent recovery, always being ready for its food, being quite lively, and showing no ill-effects from the operation. The dog was discharged from the infirmary on December 30.

AN INTERESTING CASE OF HEMIPLEGIA IN THE DOG.

BY ALICE M. CORTHORN, M.B., KENSINGTON, AND FREDERICK HOBDAY, F.R.C.V. F.R.S.E., LONDON, W.

THE patient, an Irish terrier, male, 7 years old, which was born in India and had spent three years in that country, came to England in 1902. He was apparently in the best of health until April 13, 1905, when, as he was starting across the road to interview an organ-grinder's monkey, he came into collision with a motor car, and was knocked down, being bowled over twice; he was hit violently on the left side by the front wheels, staggered to his feet, and then fell under the hind wheel, but we cannot quite say whether that passed over him or not. He got up and came back carrying the right hind-leg, which had a long flesh wound on the inner surface of the thigh. The only other feature which was perceptible was a patch of dust on the right shoulder. The wound was cleansed and sutured, the dog appearing to suffer very little discomfort. For the next two days the patient appeared to be in perfect health, but on the night of the 15th he was very restless and continually changing his position. On the 16th he lost his appetite, and from this date began distinctly to lose flesh. He had licked out almost all his sutures and the wound was gaping, but healthy in appearance, and at no time during the illness did this give rise to any anxiety; the animal, however, showed frequent signs of discomfort and often jumped up suddenly with a cry of pain.

On the 21st, after one of these sudden evidences of pain, he jumped from the sofa to the floor, and it was then noticed that the left fore-leg hung limp and that he hesitated to put his full weight upon it. When attempting to get upon a sofa or chair he would climb, instead of leaping, on it as had been his usual habit. He held his head stiffly, and hesitated to lower his neck to take food.

On the 24th he walked about three miles, and it was noticed that although he took an interest in things around and in other dogs, he appeared somewhat slow.

On the 26th he was very dull all day and refused food; when this was forced upon him he returned it.

On the 27th he was unusually quiet, although he ate his dinner as usual and retained it. When out at exercise it was noticed that he gave a short cry every now and again when the left front paw touched the ground, and on the morning of the 28th he refused to rise, and cried when moved. When placed on his feet he stood literally "all in a lump," his back curved, the tail and ears dropped, and he was extremely cold and depressed. The rectal temperature was 100·4°, and it was at this stage that medicinal treatment was commenced.

The diagnosis made was that of acute rheumatism, and 5 grains of salicylate of soda with a little acetate of ammonia and ether were given twice a day. This seemed to give a satisfactory result, and on the following day the dog was decidedly better and ate his food with relish.

FIG. 1.—Showing the paralysed condition of the fore-leg when the dog was sitting down.

April 30.—On this date we noticed extensive subconjunctival hæmorrhages; nearly the whole of the sclerotic was coloured in the right eye, and there were two large patches in the left. He was dull and lethargic, with occasional spasms of pain, the bowels and bladder acting satisfactorily.

May 1.—The medicine was now changed to aspirin, 5 grains three times a day. The patient was very dull, seemed quite unable to settle himself comfortably in any position, and the pain was very frequent. On the following day he was distinctly brighter, and even went for a fair walk in the afternoon. The right eye, however, was kept closed, the hæmorrhage beginning to take a rusty-looking colour. On the following day he was more dull, distinctly suffering pain, and scarcely able to climb upstairs. About 2 a.m., he was continually moaning, panting violently, and preferring to stand with his head pressed against something; the neck muscles were in a constant fit of quivering, and he seemed in violent pain when the hand was placed on the back of his neck.

Half a grain of morphia was injected, and this caused him to doze off quietly for a while.

Knowing that a portion of the animal's life had been spent in India, and thinking that possibly *Filaria immitis* might be present, Colonel Nunn, who had had some experience of this trouble, was called in in consultation ; he however, decided, against the presence of this worm. The dog was now put on 3 grains of iodide of potassium three times a day.

On May 5 and 6 it was noticed that the hind-legs were moved with difficulty, and were dragged along during progression. The bowels and kidneys were acting normally ; there was difficulty in mounting stairs, and a peculiar muco-purulent discharge from the right nostril ; when attempting to pass urine the dog almost fell over. On May 7, about midnight, he seemed in great pain, constantly crying, and panting heavily. Morphia injections were administered at 2 a.m. and

FIG. 2.—Showing the hemiplegic condition of both left limbs.

8 a.m. ; there was considerable discharge from the right eye and right nostril, the latter being continually plugged with crusts. In the afternoon there were definite signs of paralysis in the left fore-leg and, in the evening, of the left hind-leg, the paws being flexed and the leg dragged ; the right side of the face seemed a little more expressionless than the left.

May 8.—Paralysis now more marked ; when walking he staggered a good deal and fell over once or twice ; the right nostril was plugged with discharge. The dose of iodide was increased to 5 grains, iodide of sodium being substituted for the iodide of potassium, and the dog kept as quiet as possible in a sunny spot. Two curious, symmetrical, swellings formed in the region of the neck, one on either side, but they were not tender at all on pressure, and there was no apparent cause

for their formation. On the following day the paralysis was more marked still, the left side limbs were dropped, the paws being flexed and the body held in a bunched-up fashion. The temperature at 9 a.m. was 102·2°, and at noon 101·2°. There was no evidence of acute pain, but when walking the animal fell over several times.

May 10.—The temperature varied from 100·6° to 101.6°, the dog seemed drowsy and dull, but cried when being made to move. The swelling in the glands at the side of the neck had perceptibly subsided; there was a muco-purulent discharge from the eyes and nostrils, particularly on the right side.

A grain of calomel was administered, and the iodide of sodium, 5 grains, given four times a day. On the following day there was a slight improvement both in the condition of the eyes and in the symptoms of the paralysis, and from this date the paralysis commenced slowly to disappear and the appetite returned.

On the 14th he was able to walk some little distance, and by the 22nd, except for a certain limpness every now and again, he was able to run tolerably well; his body, however, was drawn round the left side and the back distinctly curved. He frequently stumbled going up and down stairs, and had not complete control over his limbs. He still had great difficulty in springing upon a sofa or chair, but otherwise ppeared to be going on as well as could be expected.

About six weeks after this date a distinct relapse was noticed, there being a failure in controlling the extensor muscles in both left limbs. The administration of iodide of sodium was continued for another fortnight, and improvement was again well marked. The drug was stopped for about a week, and from that time until now there has been no relapse,

January, 1906.—At the present time the dog appears in excellent health and has apparently made a satisfactory recovery.

PARALYSIS AND PERMANENT DILATATION OF THE ŒSOPHAGUS OF A HORSE.

BY F. J. POLLOCK, F.R.C.V.S., NORWICH.

THE original specimen from which this interesting photograph was made was shown to a number of veterinary surgeons in the Eastern Counties, who had met together to see a clinical demonstration of the operation of ovariotomy performed upon a troublesome mare. The patient, a cart-horse, 12 years old, was first brought under observation about a year ago. The animal presented all the symptoms of choking, a large quantity of mucus being ejected from the nostrils and, at the same time, a quantity of food was returned from the mouth. When examined from the outside it was readily seen that the œsophagus was much dilated, and upon digital examination it distinctly pitted on pressure. Little could be done in the way of treatment beyond allowing the patient to drink as much fluid as it wished. During the past twelve months there has been a recurrence of the above symptoms about every fortnight and latterly every few days.

When last attended, on November 10, the horse had dislodged, by the act of retching, fully a quarter of a pail full of masticated food, and the poor beast presented a most dejected appearance. By my

Dilation of the lower end of the œsophagus.

advice the owner had him slaughtered, and the photograph shows clearly the condition presented by the œsophagus after death.

TUMOUR ATTACHED TO THE DIAPHRAGM OF A HORSE.

BY J. NICHOLAS, M.R.C.V.S., LIEUT. A.V.D., MIDDELBURG, TRANSVAAL, SOUTH AFRICA.

THE animal, a brown gelding, 12 years old, had been under treatment since September 25 for pneumonia complicated with pleuritis, and died on November 28. On making a *post-mortem* examination the usual lesions were present, but on opening the abdomen a large tumour was noticed on the upper and right border of the

diaphragm adhering to the pillars. The growth was elongated in shape, measuring 11 ins. in length and from 3 to 5 ins. in width, and weighed 8 lbs. It was soft and inelastic in constituency, and on section the two cut surfaces much resembled those of an orange divided in two.

The contents were gelatinous in consistency, and a light straw-coloured fluid exuded from the cut surface. The tumour had a very firm, hard external coat, which was about $\frac{3}{8}$ of an inch in thickness.

AN INTERESTING CASE OF ŒSTRUM AFTER OVARIOTOMY.

BY FREDERICK HOBDAY, F.R.C.V.S., F.R.S.E., KENSINGTON.

In March, 1904, I was asked to operate upon a fox terrier puppy, 5 months old, which had never yet been in œstrum.

The operation was performed satisfactorily, and I am perfectly sure that every particle of each ovary was carefully removed. Chloroform was the anæsthetic used, and the usual regard was paid to antiseptic precautions. To all appearances it has not made the slightest difference to her sexual propensities, for the owner writes as follows: "The little bitch has to be shut up for between a fortnight or three weeks twice a year, and other dogs come about more than usually at these times; she herself displaying the usual excitement when she hears or sees them. The discharge is coloured, and she is altogether in the same state as if she had never been operated upon. This has now happened three times, viz., September, 1904, March and August of 1905. In March it lasted about a fortnight, and in August about five days. She seems perfectly well in health."

The case is interesting and merely confirms the reports of other observers that occasionally the removal of the ovaries, and even the uterus too, will not of necessity deprive the patient of sexual appetite.

In this case, too, it is particularly to be noted that the operation was performed before an œstral period had taken place.

RINDERPEST.*

BY GEORGE TURNER, M.B., MEDICAL OFFICER OF HEALTH TO THE TRANSVAAL GOVERNMENT.

As you are probably all well aware, rinderpest first appeared in these Colonies in the year 1896. It had long been endemic in the Soudan, and my friend and colleague, Mr. D. Hutcheon, the Chief Veterinary Surgeon for the Cape Colony, has shown fairly conclusively that it reached this country from that place.†

The appearance of this bovine scourge naturally caused much alarm

* A paper read before the British Association at the meeting in Johannesburg.

† *Agricultural Journal* (Cape), September 17, 1896, p. 498. I cannot consult the original official report.

in the various South African States and Colonies, and steps were immediately devised to prevent or limit the extent of its progress.

In England, in 1865, the destruction of infected herds alone proved to be of any service. The disease was imported into Hull in May, 1865, and was detected in London by Professor Simmons on the 10th of the following July. It spread rapidly.

By December 30 of that year 9,753 farms were infected and 159,710 cattle were attacked.

On May 9, 1866, an Order in Council was issued requiring the destruction of all diseased and infected animals. The result was most favourable, because in the following November there were only sixteen cases of rinderpest on eight farms. That this result was owing chiefly to the effect of slaughtering is rendered highly probable by the fact that although the disease was twice introduced into Ireland it did not spread beyond the first animals affected, and those which came in contact with them, the whole of which were promptly slaughtered.*

The Chief Veterinary Surgeon of the Cape Colony, therefore, in my humble opinion, acted judiciously when he recommended slaughter and the construction of a fence between the infected and uninfected areas, especially as Mr. George Fleming, a very high authority, wrote :—

" . . . . The endeavour to find a cure for it (rinderpest) must be considered futile, and in Africa as little else than insanity ; there is no cure for it, and even if there were how could it be applied ? The well-known Veterinary Sanitary Police measures can alone be resorted to with success. . . . ."

And again, in reply to a question addressed to him by the Agent-General, he wrote on June 17, 1896:—

" . . . . What induced me to write to the *Times* was the apprehension that the authorities in Africa might lose valuable time and neglect the only effective measures in search of a cure, a mistake which cost this country (England) millions of pounds in 1865-66 and brought about the ruin of hundreds of cattle-owners. . . . ."†

But although no doubt Mr. Hutcheon's advice was sound, he had a much more difficult problem to solve than was put before the authorities in England thirty years previously.

In the first place, the ox in South Africa is a far more important animal than at home, where its principal use is for food, and one rarely sees oxen ploughing or performing any other work.

Here it is the chief means of heavy transport. At the time the epidemic occurred the whole of the goods traffic and even the stage coaches, in places, were drawn by oxen between Mafeking and Kimberley, so that when the disease reached Bechuanaland, I have been informed by those who were eye-witnesses, that ox-waggons could be seen on the main road stranded and deserted, because the oxen had died and there was no other means of traction. Moreover, there was another and very essential difference between these circumstances in England and in South Africa. The former, thank God, is an island, and therefore it was comparatively easy to regulate, restrict, or suspend the importa-

* *Agricultural Journal* (Cape), September 17, 1896, pp. 447 and 496. I cannot consult the orignal official report.

† *Ibid.*

tion of animals. To do so in the Cape necessitated the construction of miles upon miles of expensive fencing and that the whole length of that fence should be patrolled.

Fortunately the Cape Government, also acting under Mr. Hutcheon's advice, while following his and Mr. Fleming's recommendations as to "Veterinary Sanitary Police Measures," viz., restriction of traffic and the slaughter of infected herds, was not discouraged by Mr. Fleming's prognostication as to the futility of attempting to discover a medical means of prevention and cure. The services of Dr. Koch—the well-known German bacteriologist—were obtained; nevertheless, but for the precautions taken by Mr. Hutcheon's advice (which although eventually defeated, as must necessarily occur, no quarantine cordon ever yet having proved effectual) the disease would have spread rapidly and Cape Colony would have been over-run, whereas Mr. Hutcheon managed to keep it in check long enough for Professor Koch to perform his task.

Dr. Koch went to Kimberley in the first week of December, 1896, and left Africa in the following March, having discovered and perfected the bile method of immunisation.

As Dr. Koch was compelled to return, and as it was necessary that someone in the service of the Cape Government should be thoroughly acquainted with Dr. Koch's method, his opinions as to the means and possibility of further improvements, &c., the Minister of Agriculture caused me to be temporarily relieved of my duties as Medical Officer of Health of the Cape Colony and I was sent to Kimberley where I remained with Dr. Koch for about three weeks.

During this short period Dr. Koch lost no opportunity to explain to me his process, his opinions as to the way in which it acted, the most promising directions for future experiments, and how—in his opinion—the process might be improved. The following is the result of my experiments and observations.

Signs and Symptoms of Rinderpest.

When an animal is attacked by rinderpest in the usual manner, through contact with the nasal mucus or the fæces of a sick animal, the disease usually has an incubation period of between four and five days. When the disease is conveyed by means of the inoculation of virulent blood into the subcutaneous tissues the incubation is much shortened, it is then more usually sixty hours, sometimes forty-eight hours, and in one instance the first rise of temperature was noted after an interval of only thirty-six hours.

The first sign of sickness is given by the thermometer. Normally, amongst the cattle at Kimberley, the temperature, in the rectum, was 100° to 101° F. in the morning and 101° to 102° F. in the evening; this, after the incubation, would rise suddenly to 105° or 107° F.*

Frequently, however, the onset of the fever was marked by the fact that there was no morning remission of temperature, the thermometer showing the same number of degrees as in the preceding evening. Very rarely a decided fall ushers in the specific fever.

For a few days after the onset of the fever the animal rarely shows

* In districts in which redwater and the disease known as *gal ziekte*, or bovine malaria, is common, the daily fluctuations are often much wider than 1° or 2° F.

any symptoms of ill-health, it eats and chews the cud as usual, and its condition would not attract attention unless the thermometer was used.

The first noticeable symptoms are that the animal ceases to eat and does not chew the cud, the coat becomes rough, the nose dry, and it frequently grinds its teeth.

After about four days' fever more specific symptoms are apparent. There is running from the eyes and nose, the conjunctivæ are injected, diarrhœa sets in, the fæces become offensive and often contain blood, the animal loses weight, when standing its head and ears are dropped, but it frequently lies down. Often a peculiar tremor, especially of the muscles of the hind-limbs, is observable.

About the seventh day of the fever, sometimes later, the breathing becomes rapid, the beast is restless, and then a sudden fall of temperature is noticed, which is usually followed by death in a very few hours.

If, instead of a sudden fall, the temperature is lowered gradually the outlook is more favourable, and the animal may recover.

Recovery is usually a prolonged process, because a secondary fever, due to septic infection, usually occurs, which carries off most of those which survive the specific fever.

If the nasal and buccal mucous membranes are examined, the former is almost universally found to be deeply coloured; the papillæ of the latter are frequently in a similar condition, and aphthous ulcers may be found on the gums.

On the Kimberley * Station the ulcerations in the buccal mucous membrane, just mentioned, were quite the exception; at Pretoria, on the other hand, they were commonly found.†

In a few instances the subcutaneous tissue over the loins was emphysematous and crepitated very distinctly under pressure; but this only occurred during the secondary fever.

Post-mortem Appearances.

When *post mortem* is made, if the animal dies or is killed before the occurrence of the secondary fever, the lungs, heart, liver, kidneys and spleen are in their normal condition, with the exception that small subserous extravasations of blood are often met with, usually between the ventricles or between the ventricles and auricles.

The intestinal tract is affected from the fourth stomach to the anus.

I have never met with any lesions in the first three stomachs, but the mucous membrane of the fourth is always affected. It is inflamed, often a deep plum colour, spotted here and there with red petechiæ which are denuded of epithelium; there are often ulcers, especially in the vicinity of the pylorus.

In the majority of instances the whole of the intestines, small and great, are inflamed; but there are one or two where inflammation is more likely to be met with than elsewhere.

These places are just beyond the opening of the gall-duct, a place 18 ins. or 2 ft. beyond the ileo-cæcal valve and again in the rectum. Occasionally the inflammation is confined to these localities.

At Kimberley ulcers were common in the rectum, but I am inclined to believe that these were due to rough usage with the thermometer, because I did not meet with any in Pretoria.

* 1896-98. † 1901-02.

Peyer's patches are thickened, denuded of epithelium, dotted with red spots, and occasionally ulcerated.

The gut may contain a complete cast, but this was very exceptional.

The naked-eye appearances produced by inflammation in the different parts of the intestine vary considerably, and the variation depends upon the distribution of the blood supply and the folds of the mucous membrane.

Thus in the small intestine it is striped transversely and we used to liken it to the marks on a zebra; but in the colon and rectum the bowel looks as if it had been scraped longitudinally with a wide-toothed comb.

The mesenteric glands are always enlarged and resemble a string of sausages.

Although the liver, excluding the cases of secondary infection, is normal, the gall-bladder, with few exceptions, is inflamed, the mucous membrane is thickened and presents numerous petechiæ, indeed I have seen the contents of the gall-bladder to consist almost entirely of blood.

The internal surface of the urinary bladder presents an appearance similar to that just described, but it is usually not so evident; the urine in the majority of cases is of a normal colour.

The symptoms and appearances during and after life indicate an attack of an acute infectious fever, with which a specific organism is invariably associated.

Until recently I should not have hesitated to state that any animal presenting these *post-mortem* appearances had died of rinderpest. The opinion would, in the majority of instances, be correct. There is an exception, however, of the greatest importance in South Africa, north of the Orange River.

In August, 1902, the Resident Magistrate in the Barberton District sent to me for serum for use amongst a herd of cattle sent by the Government to Komati Poort. Fortunately I received some information as to the dates of infection, the duration of the fatal illness, and the comparative slowness with which the disease had spread, which induced me to doubt the correctness of the diagnosis.

I refused to forward any serum to Komati Poort until I had had an opportunity to make some *post-mortem* examinations.

Accompanied by my friend and colleague, Dr. Theiler, I visited the place and we were able to make a very large number of *post mortems*. To the naked eye the pathological signs were those of rinderpest, excepting that the liver and spleen were enlarged, and the subperitoneal fat was jaundiced, but the microscope disclosed that the disease with which we had to deal was Rhodesian tick fever.

In countries where that latest of South African bovine scourges is met with no *post-mortem* diagnosis is beyond dispute until a microscopical examination of the blood has been made.

Had I sent the serum it would have been used, it would certainly have produced no effect whatever, and doubts would have been cast on its efficiency.

Dr. Koch diligently searched for this organism in the blood and the nasal and intestinal mucus, but without success.

Many observers in Europe and Asia have asserted that they have seen and cultivated the rinderpest organism. The latest claimants,

to my knowledge, being Messieurs M. Nencke, N. Sieber, and W. Wyznikiewiez.* They published a paper with photographs and drawings, and described a microbe to the presence of which in the blood they attributed rinderpest.

These objects are not contained in the blood of animals suffering from rinderpest in South Africa.

The methods these observers employed to isolate and cultivate the organisms will not stand fair criticism. The organisms resemble amœbæ and were said to have been cultivated in a fluid medium, which necessarily offers numerous facilities for errors.

The plates show evidence of faulty technique in preparation and in photography.

The experiments described by those gentlemen have since been repeated at the Rinderpest Station, Pretoria, but their results have not been confirmed.

I have examined the blood on hundreds of occasions, using every method with which I was acquainted or could imagine, or which I met with in various text-books, but without success.

I have examined the lymph from the ductus communis, and from enlarged glands, with no better success.

That there is an organism is demonstrable, because if serum, slightly red with suspended corpuscles, be allowed to stand long enough to admit of subsidence, or if the fluid be centrifugalised, then the top, clear, supernatant serum can be injected into all animals with impunity, while the red deposit as surely conveys the disease.

Again, if the red cells contained in defibrinated blood be destroyed by any suitable saline solution and the mixture be centrifugalised, then the red liquid is harmless, whilst a very small quantity of the white jellyfish-like deposit is found to be virulent.

The organism must be in this white deposit, yet I have examined it frequently and by many methods, but was never successful in my search. Incidentally, I may suggest that it is probably intracorpuscular, but, as I shall show later, must be of sufficient size to be visible by means of a one-twelfth lens.

Dr. Koch demonstrated conclusively that the rinderpest organism was easily destroyed by the action of light and air, especially the latter. This observation has been confirmed by Captain Leonard Rogers, I.M.S.† He found that the dried hides were not infectious, but on the other hand, that the marrow of unbroken bones remained virulent for one month while exposed to a temperature between 37° and 60° F.‡

Dr. Koch showed that 5 per cent. of carbolic acid destroyed the virulence of rinderpest blood, and that glycerine had a similar effect.

This was a preliminary investigation, undertaken because it was of great practical importance. There is a considerable over-sea traffic in hides, and it was probable this would be stopped if the disease could be conveyed by them, and in view of the fact that thousands of animals were dying of the disease, it was important to know that dried hides taken from such animals were harmless.

* *Centralblatt für Bacteriologie, Parasitenkunde und Infections krankheiten*, March 1, 1898.

† "Investigation in the Method of Inoculation against Rinderpest." *Imperial Bacteriological Laboratory*, 1898-99, p. 16, para. 31.

‡ *Ibid.*, p. 20.

Professor Koch, as was natural, first turned his attention to the possibility of producing a serum which was either preventive or curative, but eventually he devoted the greater part of his attention to the perfection of the bile method.

I have seen it stated that he got the idea from the natives. This is not so. He himself told me: " . . . . I was testing the virulence of the various fluids of an animal dead of rinderpest, and amongst others used gall from an animal which had died of rinderpest, fully expecting that it would communicate the disease. I injected 10 cc. into an animal, and it was none the worse; on the contrary, when it was subsequently inoculated with rinderpest blood it resisted its action. I immediately tested the bile from six animals and obtained a similar result. . . . . "

Thus was the bile method which has proved of such signal service to South Africa discovered.

The immunity conferred was evidently an active immunity, due to the presence and action of a living organism, because when four animals were infected with 10 cc. of the same sample of rinderpest bile, and then two, four, six and eight days later with virulent blood, those infected on the second and fourth day after the bile injection had fever and the first died. The other two escaped. Since then experiments show that immunity is not always complete even on the sixth day.

The bile can be injected anywhere into the subcutaneous tissue, but it is best to do so into the dewlap where the skin is loose, because a hard, brawny swelling arises at the seat of inoculation; the animal n consequence becomes stiff, and may even be unable to walk, but, unless the bile has been contaminated with pyogenic organisms, the swelling does not suppurate and gradually subsides.

I am under the impression that if suppuration occurs, in some cases at least, the immunity is not so complete.

Except for the stiffness, animals do not exhibit any characteristic symptoms, there is no fever, and the secretion of milk is not interfered with.

Dr. Koch's opinion as to the way in which bile from a rinderpest animal acted was as follows:—

The bile contains the organism in full activity, but its activity is restrained by the presence in the bile of a chemical substance, probably of an albuminoid nature. The effect of the bile is principally local, but at the same time it affects the whole system in the same way as vaccine lymph does in respect to variola.

Dr. Koch based his opinion upon the following experiments:—

If 5 per cent. of phenol be added to rinderpest blood, and be allowed to stand twenty-four hours, the animal injected with such a mixture is not protected.

But if to a sample of bile in which the rinderpest organism has been destroyed by phenol, rinderpest blood be added, and injected at once, the protective power of the bile is restored and the animals injected with such a mixture are immune.

Dr. Koch concluded that he had first by the action of the phenol removed the original organism contained in the bile and had replaced it by the virulent organism contained in the blood, and as the latter was known to be virulent, he concluded that, as the effects were similar, the first was also virulent.

I received this opinion directly from Dr. Koch, wrote it down in

my notebook, and read it over to him to be sure my notes were correct.

After thinking the matter over carefully, I ventured to doubt the correctness of the opinion I had received and tested the truth in the following manner:—

First, I put rinderpest gall into a Berkefeld filter and by means of an air-pump drew the fluid part into a receiver. The receiver was then removed and a second substituted. The filter was then washed twice by passing sterile salt through it to ensure the removal of all fluid and soluble matters. The Berkefeld filter was then surrounded with normal salt solution and scraped with a sterile platinum spatula to wash off any matter adhering to it. The fluid was collected in a test tube and examined under the microscope. Nothing could be seen but here and there a stray coccus which had been contained in the original bile. The filtrate was then injected into an ox (No. 204).*

The injection apparently was absolutely without any effect. The animal ate well, had no symptoms, and its temperature was absolutely normal. After ten days had elapsed it was injected with 0·2 cc. of virulent blood and found to be immune.

Ten cc. of the filtered bile were injected into each of four other beasts. These animals likewise exhibited no symptoms and experienced no rise of temperature, but on the tenth day, when they were injected with the same dose of the same virulent blood they all had the disease seriously and two of them died.

I have repeated this experiment on several occasions with precisely similar results.†

This, in my opinion, tends to prove that the organism contained in rinderpest gall is a modified organism, it is impossible to suppose that, if it had not been modified, the subject of experiment would have escaped after the injection of such a big dose of infecting material.

It is also conclusive evidence that the organism, whatever its nature, must be of such a size as to be visible under the microscope or it would have passed a Berkefeld filter.

I tested the question in another way. If the organisms in the bile were virulent organisms restrained by a chemical substance in the bile, then the injection of 10 cc. of rinderpest gall directly into the venous circulation would probably have the effect of diluting the chemical substance to such an extent that the organisms would be little effected by it.

I laid bare the jugular vein of an ox and introduced the needle of a Koch syringe which had been filled with normal salt solution. The salt was pumped into the circulation until it reached the zero mark (= 10 cc.), and then 10 cc. of rinderpest gall were introduced into the syringe, this, too, was driven into the vein, and, lastly, the syringe was washed twice with normal salt solution. The wound was washed with a disinfectant and sewn up with carbolised catgut. The animal

* This ox was the first prepared for the manufacture of strong serum. He eventually provided blood which was sold for £200.

† Dr. Kolle has stated that when rinderpest gall was centrifugalised, the residue washed, to remove all the fluid parts, that the resulting particulate matter is followed by rinderpest. But this experiment was undertaken when the Kimberley Station was highly infected. We are no longer depending on gall for immunity, and an outbreak of rinderpest was of no consequence on the station, therefore the stringent and irksome precautions which had been taken previously were no longer enforced, and we often had cases of natural infection.

never had a bad symptom, but it was not immune, because it subsequently succumbed to 0·2 cc. of virulent blood.

Experiments show that bile during the first three or four days of the disease has little protective power, and Professor Koch recommended that it should be taken on or after the seventh day.

He further recommended that the bile selected for immunisation should have a bright green colour and show, when shaken, a white froth and be free from smell.

However, experience has shown that bile of colours are frequently just as useful, provided they are clear and do not smell.

It has been stated that it requires the death of seven animals to procure sufficient gall for the immunisation of 100 beasts, but in my experience the number was more nearly three or four if the animals were slaughtered about the seventh day, after the rise of temperature and before the animal was collapsed.

All green galls are not protective. I spent much time endeavouring to find some method of recognising those which would prove effectual, and I am of opinion that only biles which give the oxyhæmoglobin band under the spectroscope, or preferably those which give the methæmoglobin band, or in conjunction with the oxyhæmoglobin bands, are effectual.

My opinion of the manner in which the bile of a rinderpest animal acquires the property of conferring immunity is as follows :—

The mucous membrane of the gall-bladder of an animal suffering from rinderpest is always inflamed. There are small petechiæ in the course of the veins, the blood continually enters the bile in small quantities.

Under the conditions which obtain *in vitro* the bile would destroy the virus, but as that which enters the bladder has been for some time confined in the immediate neighbourhood of the gall, and in liquids gradually more and more impregnated with gall, it has become habituated by degrees to live in a fluid containing bile, and in so doing has become attenuated.

The substances which thus act in the organism are probably the bile salts.

I prepared some pure bile salts from normal gall in the usual way, made a 5 per cent. solution, and added to the solution 10 per cent. of virulent blood.

If used in 10 cc. doses soon after mixture the animal had the disease, but if allowed to stand for twenty-four hours the animal generally escaped.

I have even immunised an animal by the action of bile salts in the virulent organism at 37° F. for 116 hours.

These experiments were undertaken with the object of discovering a means of manufacturing a modified organism artificially by the action of bile on rinderpest blood, but as by this time I had managed to produce an effective serum they were abandoned.

When first Professor Koch demonstrated the novel fact that rinderpest gall was an immunising agent, those fond of theorising surmised that the gall itself or the animals subjected to its influence might convey the disease.

This fear acted on those in authority in such a way as to lead to restricting the use of bile amongst herds actually infected or in the immediate neighbourhood of infection.

That an animal undergoing immunisation by means of gall was not dangerous to others is sufficiently proved by the fact that blood from such beasts was repeatedly injected into healthy animals without producing any effect whatever.

During the time that the station at Kimberley was conducted under conditions intended to rigorously exclude natural infection, we did not meet with any case of rinderpest following the inoculation of rinderpest gall; during that period over 200 animals had been injected with many specimens of gall, at least 100, but rinderpest was in and around Kimberley, and when animals already infected were delivered to the Station for experimental purposes, injection was occasionally followed by the disease.

The various inoculators, however, sent in numerous reports tending to show that bile inoculation was followed by rinderpest, but these reports will not stand fair criticism.

There is often evidence that the herd was already infected before the gall was used: thus one herd of 133 was inoculated on May 9, 1897; it is stated that none of the animals were infected. The whole herd sickened and 100 died, but there is a note stating that a cow died on May 8.*

Again, there are numerous instances in which the same sample of bile (mixed) was used in more than one herd, and in which one herd suffered and the other or others escaped. In such circumstances we have three factors, viz., the herd, the bile, and the operator; the last two are constant, the first variable, but by some process of reasoning the variation in the results is attributed to one of the constants, and the variable is neglected. This is incomprehensible to me.†

In the *Agricultural Journal* (Cape), August, 1897, p. 175, there is an account of the results obtained from the inoculation of 634 animals; of these 506 were members of five infected herds, and the number which survived after inoculation was 49, or 9·6 per cent. The remaining 1,128 belonged to four herds said to be free from infection; of these 1,114, or 98·6 per cent., survived. I had only one opportunity to experiment on a herd which was free from any probability of infection. I had repeatedly asked to be allowed to operate on clean animals on one of the islands off the coast, where, if rinderpest resulted, it would be of no practical importance, but I could never obtain permission to do so. At length Dr. Smartt, of Britstown, placed a herd of 160 at my disposal. Rinderpest was not known within forty miles. The animals all received 10 cc. of bile, and not one subsequently sickened.

Even in this case the chance of unknown infection was not so remote as was supposed, because within eight or ten days the disease had appeared in a herd ten miles further from the nearest known seat of infection. Had the experiment been delayed a few days, cases might very probably have followed inoculation in Dr. Smartt's herd, and would, of course, have been attributed to the inoculation, because, as the bile does not protect until after the expiration of eight or ten days, infected cattle in the pre-febrile stage would inevitably sicken.

* "Rinderpest, its Pathology and the Means Used to Combat its Invasion of South Africa," by George Turner. *South African Medical Journal*, November, 1897.

† *Idem.*

Dr. Koch's method no doubt proved most valuable to the Colony, and would have rendered more service if it had been carried out in the way in which he intended it should be performed, *i.e.*, to commence inoculating in the clean zone towards the infected area, so as to provide a belt of immune cattle.

Certain drawbacks are connected with the bile process—nearly all processes have drawbacks—they are :—

(*a*) We must face a loss for the provision of bile of between 4 and 7 per cent. per 100.

(*b*) The bile is not in any sense curative.

(*c*) It does not protect completely until after the expiration of from six to ten days, thus leaving a loophole for subsequent infections.

(*d*) The immunity is short.

(*e*) The bile will not keep.

It has one great advantage, it can be carried out wherever and whenever rinderpest appears by any person of ordinary intelligence and without any complicated and expensive plant.

It is difficult, if not impossible, to state in figures what amount of success attended the process. On the Kimberley Station it amounted to 100 per cent. Operating on the farms in clean herds consisting of 378 animals, one only died after injection, 0·26 per cent.

The public inoculators operating on herds not dangerously infected lost 2 per cent.

Sir Godfrey Lagden informs us that in Basutoland, a highly infected area, out of 100,000 animals injected 70,000 have been saved.

In consequence of the widely spread opinion that Koch's method was dangerous, means were sought to do away with the supposed danger, and Dr. Edington advocated the addition of glycerine to gall in the proportion of one part of glycerine to two of bile. He claimed for his mixture three advantages :—

(*a*) That the disease cannot be spread by the use of glycerinated gall.

(*b*) That an economy is effected, as all galls can be utilised.

(*c*) That the mixture will keep.

The first and third propositions may be conceded ; since pure gall cannot give rise to rinderpest the addition of glycerine can obviously not do so, and the addition of 33 per cent. of glycerine does prevent putrefaction.

With regard to the second, however, it is different. The dose had to be so much increased that no economy resulted.

Dr. Edington directed that ten days after the injection of the glycerinated bile the animals should be inoculated with 0·2 cc. of virulent blood.

This process was tried experimentally at Kimberley and resulted in a loss of 70 per cent.

Dr. Leonard Rogers tried it amongst the Indian hill cattle, but without success.*

Koch, long before Edington, had experimented with glycerine and had given up the process as useless. The addition of glycerine to bile would occur to any one acquainted with its use in the preparation of vaccine lymph. Unfortunately it destroys the organism, and the power of the bile then depends on the presence in it of a small amount

* "Report on an Experimental Investigation on Methods of Immunisation against Rinderpest." Cal., 1898-99, p. 9.

of chemical immunising substance and not on the action of the microbe. As the quantity of this substance varies very much in animals the use of glycerinated gall and blood is sometimes very successful and at others simply disastrous.

In fact, in the third congress held at Cape Town in June, 1898, at which Dr. Hutcheon, Dr. Edington, Dr. Kolle and I were present, the glycerinated bile method was not even mentioned.

I was therefore surprised that when rinderpest recurred in 1901, under the advice of Dr. Edington, the use of this mixture was again recommended, and in Natal reports by veterinary surgeons supporting the use of this method and decrying the serum method were published. These reports disclose the fact that the gentlemen had very confused ideas as to the employment of serum, and, moreover, had never used a single cubic centimetre of the serum they decried. The best proof, however, that they were mistaken is that after the glycerine bile had been employed the Natal Government purchased thousands of doses of serum from the Rinderpest Station at Pretoria.

During the last epizootic (1901-2), while the use of glycerinated bile was recommended the process was materially modified.

The subsequent injection of small doses of virulent blood was given up; experience had shown it was too dangerous.

Instead, the operator was directed to make a second injection, but in this instance it was to consist of unmixed rinderpest bile.

How the idea originated it is impossible to say. It cannot have been based on experiments, or at least on any considerable number of experiments.

The Serum Method.

Dr. Theiler and Mr. Watkins Pitchford* in Natal has succeeded in immunising animals with defibrinated blood drawn from animals which had recovered, and this method was elaborated later by Messrs. Danyz, Bordet, and Theiler in the Transvaal.

The method devised by these gentlemen was as follows:—

An animal salted naturally or by the use of bile is subsequently injected subcutaneously with 100 cc. of virulent blood. When all reaction had ceased and when the animal was in its normal health it was bled, the blood defibrinated and used as follows: 100 cc. are injected into a healthy animal subcutaneously and its nose is then smeared with virulent mucus or fæces, and it is placed with animals suffering from the disease.

Usually the beast experimented on had rinderpest, and it was then again injected with 100 cc. of immunising blood.

In this way a very large number of animals were salted in the Transvaal and Natal.

Koch had experimented with the serum of a naturally salted animal, and showed that 100 cc. conferred an immunity which reached its height in a few hours and rapidly wore off.

He also attempted to increase the strength of the serum by addition of a small quantity of virulent blood.

But as the bile method at that time promised to yield more immediate results he directed his attention to it.

* I regret I am unable to do full justice to the work of Mr. Watkins Pitchford. He did not publish his results, and when I wrote to him for information he told me that his notes had been destroyed by fire.

My colleague, Dr. Kolle, and I made continuous attempts to produce a serum of higher potency than that yielded by animals naturally immune or which had been fortified by a subsequent injection of 100 cc. of virulent blood; this we were able to do in June, 1897, by the following method:—

An animal which had been salted by the use of bile was injected with 10, 50, 100, 200, 500, 700, 1,000, 1,500, and 3,000 cc., reaching sometimes 4,000 cc. (or 4 litres), of virulent blood.

After each injection the animal usually showed a rise of temperature which varied much as to intensity and duration, but this was the only indication that the animal was not in its normal condition. A few days after the animal had regained its normal condition another injection was made in a similar manner, increasing the dose of blood gradually at each operation.

I have stated that the animal should be one immunised by the bile method, but, of course, when serum has once been obtained the immunity is procured by its means; but in any case animals which have salted naturally should not be used for the purpose of preparing serum. The natural fever has so damaged their constitution that the majority cannot stand against the fortification, they do not take rinderpest, but they die, and even if they escape, the serum they yield is, in my experience, very weak.

At first we had to employ defibrinated blood to a large extent because we were unable to manufacture serum on more than an experimental scale, but we soon had evidence that the animals prepared as described yielded blood which had a much higher immunising property than any which had as yet been produced.

An opportunity soon offered itself at Niekerk's Hoop, in the Orange River Colony. On that farm there were ninety-nine animals, the remainder of a herd of 482, in which after the inoculation of bile 91 per cent. had been saved, the rest having been slaughtered. In July the immunity began to wear off, the animals sickened and several died; the disease was supposed to be *gal ziekte*.

The day we visited the farms sixteen animals were ill; we diagnosed rinderpest and our diagnoses were confirmed by inoculation. All animals received 30 cc. of immunising defibrinated blood, and though one sickened the following day the whole ninety-two were saved.

During the next two months we inoculated altogether 1,141 on several farms and succeeded in saving 1,040, or 92 per cent. The amount of success obtained differed considerably; it varied from 100 in two instances to 84 per cent., but in the instance in which the loss amounted to 16 per cent. the herd originally consisted of 472 animals and had been reduced by 160 deaths to 306.

We proved at the Kimberley Station that success depended upon early treatment. We experimented on forty-seven animals, injecting them with 20 cc. of serum from the first day of fever until the ninth. We saved 27, or 57 per cent. But eleven treated on the first day and four on the second all recovered; of seven injected on the third day five were saved, *i.e.*, a loss of only 9 per cent., while the saving during the first two days was 100 per cent.

Even on the fourth day of fever of nine beasts so treated four recovered, but after that date the serum was of little use.

I must add that these experimental animals only received a single curative injection and that a small one. I have known recovery take

place much later than the fourth day when considerable doses—100-200 cc.—were employed. I am convinced from experience that when serum is to be used at a late stage of the disease, it is better to use at once a big dose than to give smaller doses repeatedly; 200 cc. injected at once is far more effective than five doses of 40 cc. in three or four days.

At first we were compelled to issue defibrinated blood, and as the weather was beginning to be warm we experienced much difficulty in transporting it long distances, as, for instance, from Kimberley to Graaff-Reinet. We delayed bleeding to the last minute, collected the blood in well-sterilised vessels and took every precaution available, such as packing the bottles in ice, yet in several instances, when the journey occupied several days the blood when it arrived was unfit for use.

That the serum can be usefully employed by persons of ordinary intelligence, but without special training, was proved, because between July 20 and October 20, 1897, 3,318 beasts were treated by farmers and others, of these 1,077 were known to have been sick of rinderpest, yet 2,857, or 86 per cent., were saved.*

* Herds Naturally Infected with Rinderpest and Treated with Serum. All the Figures are given without any Selection.

Date	Name of place	Number inoculated	Number ill before inoculation	Number which died	Number which recovered	Percentage of recoveries
July 20	Boshof	92	17	0	92	100·0
,, 29	Alice	24	24	1	23	95·8
Aug. 19	Kimberley ...	76	60	7	69	90·8
,, 19	,, ...	188	—	25	163	86·8
,, 23	Cathcart	1,100	39	25	85	77·7
,, 23	Graaff-Reinet ...	24	24	1	23	95·8
,, 25	Kimberley ...	376	—	16	360	95·7
,, 25	,, ...	306	—	46	260	84·9
,, 25	,, ...	39	39	0	39	100·0
,, 25	,, ...	64	64	7	57	89·0
,, 28	Beaufort West ...	30	—	2	28	93·3
,, 30	Richmond ...	31	5	2	29	93·5
,, 31	Graaff-Reinet ...	15	12	3	12	80·0
Sept. 2	Aberdeen ...	150	150	35	115	76·6
,, 3	Cathcart	190	42	1	189	99·4
,, 9	Britstown... ...	37	37	6	31	83·8
,, 9	,, ...	206	—	0	206	100·0
,, 9	Middelburg ...	40	21	13	27	67·5
,, 18	,, ...	171	38	25	146	86·3
,, 18	Colesberg ...	57	57	28	29	50·8
,, 19	Graaff-Reinet ...	66	33	1	65	98·5
,, 20	Middelburg ...	5	5	2	3	60·0
,, 21	Herbert	26	26	20	20	76·9
,, 25	Graaff-Reinet ...	18	—	1	17	94·4
Oct. 5	Kenhardt	350	170	50	300	85·7
,, 10	Graaff-Reinet ...	15	12	0	15	100·0
,, 12	Piquetberg Road...	123	123	110	13	10·5
,, 12	Uniondale ...	79	42	30	49	62·0
,, 12	,, ...	243	7	0	243	100·0
,, 13	Stutterheim ...	22	14	8	14	63·6
,, 18	Griquatown ...	9	9	2	7	77·7
,, 18	Uniondale ...	128	2	0	128	100·0
,, 20	Graaff-Reinet ...	2	2	2	0	0·0
		3,318	1,077	455	2,857	86·1

It was early recognised that the injection of small doses of 20 or 30 cc. of serum only conferred immunity for a short period, such as two or three weeks, perhaps a month, but later it was found that by the use of bigger doses a much higher protection was afforded.

On October 5, 1897, an animal (No. 783) received 200 cc. of serum; on October 5, sixty-one days later, it was injected with 5 cc. of rinderpest blood without effect; on January 7 (thirty-three days) it again received 5 cc. of virulent, also without result. On February 23, *i.e.*, after another interval of forty-four days, it was injected with 50 cc. of virulent blood. It only experienced the slight rise occasioned by such an injection in an immune animal, and though left for months with infected animals it never sickened of the disease.

On November 15 an ox (No. 910) received 400 cc. of serum, and was exposed to infection by natural means. It died on January 5, fifty-one days after injection, but the *post mortem* showed that the disease was *gal ziekte*, not rinderpest.

On November 15 an animal (No. 211) was injected with 200 cc. of serum, and after a month with 5 cc. of rinderpest blood; finally on November 19, Nos. 926 and 936 each received 200 cc. of blood, and on January 7, forty-nine days later, they were tested in a similar manner. These animals also resisted infection, and did not sicken; afterwards, though exposed to natural infection for months, they remained immune.

This is conclusive evidence of two things; first, that the big dose of serum (200 cc.) will afford immunity for some two months or more; and secondly, though doubtless the subsequent injection of virulent blood made the immunity longer, that prolonged immunity was not effected by an attack of fever recognisable by ordinary means.

Amongst the various disadvantages attached to the use of defibrinated blood the following were soon recognised:—

(*a*) The fact, already mentioned, that it would not keep.

(*b*) And as a corollary it was impossible to ascertain the immunising power of any sample of such blood.

(*c*) That the injection of blood, and especially massive doses, was frequently followed by redwater, *gal ziekte* and other fatal diseases.

None of these inconveniences are associated with the use of serum only.

The following is the method by which the serum was produced:—

After an animal had been properly fortified, that is, after it had received not less than 1,000 cc. of virulent blood, it was bled.

The beast was cast, its four legs tied together, a block of wood was placed under the neck to make it taut, and a cord or band round its neck was tightened sufficiently to make the external jugular vein stand out. The skin was then carefully shaved over the vein, washed with soap, the soap washed off with carbolic lotion, and the skin swabbed with alcohol. A small incision was then made in the skin and the vein exposed. A short cannula, the diameter of a lead pencil, and about four inches long, to which a length of gutta-percha tubing was attached, was then thrust into the vein.

This cannula and tube had previously been disinfected in the steam steriliser, and were then kept in absolute alcohol until they were required for use, but immediately before use they were put into carbolic lotion, from which they were taken to be inserted into the animal.

A few cc. of blood were allowed to escape, and then blood to the extent of about four litres—the amount depended on the size of the beast operated on—was received into a Loefler's flask.

This Loefler's flask had been prepared as follows: An inch or two of boiled water was put in the bottom of each flask, the necks and side tubes plugged with cotton-wool, and they were sterilised twice in the steam steriliser.

When used the plug in the neck was withdrawn, the water poured out, the rubber tube from the cannula inserted, and the wool-plug replaced.

The flasks when filled were placed in a cool room until coagulation had occurred and were then put in the ice room.

After twenty-four hours the separated serum was poured off, the quantity noted, and 0·5 per cent. of phenol dissolved in as little water as possible added to the serum.

Again, twenty-four hours later more serum was taken and treated in the same way.

Serum thus prepared will keep indefinitely, and at first at Kimberley, and during the whole of the Pretoria epidemic, it was made in this manner, but at Kimberley, after the first few months, a centrifugal machine capable of dealing with 10 litres at a time was used to render the fluid perfectly clear.

The method we finally adopted to procure a prolonged immunity was as follows:—

The animal was injected with serum on one side of the body, and with a small dose of virulent blood on the other.

If the quantity of serum administered was properly regulated the animal showed a slight rise of temperature on or about the fourth day after the injection, this continued for a few days, but there were no other symptoms.

If the quantity of serum used was too great the beast did not experience any fever, on the other hand, if it were too small the animal might go through various degrees of rinderpest, from fever without other symptoms to a well-marked and fatal attack, according to the amount of the serum employed.

It was evident that to carry out the experiment successfully it was necessary to know, fairly accurately, the strength of the serum to be used; and to arrive at this knowledge it was first necessary to know the weight of the animal.

The serum was treated in the following way: When a large carboy full had been collected, ten animals were selected. Two were injected with 10, two with 15, two with 20, two with 30 cc. each, and each animal was simultaneously inoculated with a small quantity of virulent blood.

The size of the animals had also to be considered. We based our experiments on the supposition that 600 lbs. live weight was about the average. Thus if an animal only weighed 400 lbs. a dose of 13·3 cc. was considered equivalent to 20 cc., and conversely if its weight was 800 lbs. live weight then 26·6 cc. were given instead of 20 cc.

The animals were tied up, provided with fodder and water, their temperatures were taken twice daily, and the result of the experiment noted.

If those injected with 10 cc. died, the dose was obviously too small; if those treated with 15 cc. were severely ill the same conclusion was arrived at. If, as usually happened, those which had received 20 cc. only experienced a slight rise of temperature, and those with 25 and 30 cc. none, then it was considered that 20 cc. was the appropriate dose for animals of 600 lbs. weight.

I say that usually 20 cc. was proved to be the correct dose for an average animal, because as huge quantities of serum, drawn from a hundred or more animals, were tested, and the serum was often drawn from the same animals its strength was very constant, but samples were found which succeeded best when only 15 cc. were employed, and one or two of which the proper dose was 30 cc.*

At first it was supposed that it would also be necessary to regulate the amount of infecting blood, but it was found by experiment that no more fever was produced by the inoculation of 0·2 cc. than of 200 cc. of virulent blood after the injection of 20 cc. of immunising serum; in fact, Dr. Nicol, of Constantinople, has prepared animals for the production of serum by the simultaneous injection of 25 cc. of serum and 4,000 of virulent blood.

It was very difficult to obtain information as to the results of inoculations performed by farmers and others. Forms were distributed with each consignment of serum sent out, and farmers were begged to fill them in. The majority never took the trouble to reply, while some who replied made returns which were obviously incorrect. No less than seventy-six imperial gallons were sent to Cape Town, but we have next to no reliable information as to the results obtained.

Still I have authentic information concerning 10,407 animals inoculated, principally by persons in no way connected with either the Kimberley or Pretoria Stations. The loss was only 1·3 per cent. The worst result, apparently, was obtained at the Kimberley Station, which showed a loss of 36·1 per cent.; but it must be remembered that Kimberley was essentially an experimental station, and that many

* It was impossible to actually weigh each animal, and therefore I made experiments on Robben Island, from which I deduced the following formula:—

Let G represent the girth of the animal behind the shoulders in feet and decimals of a foot.

Let L be the length taken along the back from a line drawn vertically from the front of the shoulder to one drawn vertically through the hambone, also in feet and decimals of a foot.

Let W be the weight in pounds.

Then $G^2 \times L \div 0{\cdot}142 = W$.

Dr. Leonard Rogers, in India, uses the same formula, but his constant is 0·12. This is due presumably to the different formation of the African and Indian breeds. The two calculations applied to the same measurements produce very different results. For instance, if an animal had a girth of 5 feet and a length of 3 feet, then by the Kimberley formula:—

$$\frac{G^2 \times L}{0{\cdot}142} = 528 \text{ lbs.}$$

By the Indian:—

$$\frac{G^2 \times L}{0{\cdot}12} = 625 \text{ lbs.}$$

animals died during the process of treating the serum, and that doses as low as 5 cc. were sometimes exhibited.*

Dr. Leonard Rogers† operated in India on two different kinds of animals, the one came from the plains and the other from the hills.

Experimenting with twenty-two cattle from the plains after they had received from 6·0 cc. to 32·8 cc.‡ of serum and had been injected with blood, he says that nine showed good temperature reactions with

* Results Obtained by Simultaneous Inoculations.

Name of the farm or station where inoculation was performed, or name of one farmer to whom the cattle belong	Number of cattle inoculated	Number of cattle which died consequently from rinderpest	Percentage	Number of animals which recovered	Percentage	Doses of serum used, and remarks
1. Rhodesia.						c.c.
Buluwayo Station ...	1,909	0	0	1,900	100·0	15-25
Gwelo Station	122	0	0	122	100·0	
Ramaquabaan Station...	1,110	25	2·2	1,085	97·8	
2. Cape Colony.						
(i.) Upington District—						
Mr. Turner ...	272	2	0·8	270	99·2	20
Mr. Moller ...	2,200	15	0·7	2,185	99·3	
Mr. Harris ...	1,100	5	0·5	1,095	99·5	
Mr. Steyn... ...	1,500	4	0·3	1,496	99·7	
(ii.) Herds inoculated—						20-40
Beaconsfield ...	22	0	0	22	100·0	
Griqualand West ...	37	0	0	37	100·0	
Kimberley... ...	27	1	3·7	26	96·3	
Jacobsdal	52	2	3·8	50	96·2	
Belmont	7	1	14·3	6	85·7	
Zwaartkop ...	80	3	3·7	77	96·3	
Groot Druck ...	80	0	0	80	100·0	
Uniondale ...	84	0	0	84	100·0	
,, ...	89	0	0	80	100·0	
Mr. Oakley ...	4	0	0	4	100·0	
,, ...	61	0	0	0	100·0	
	8,756	58	0·6	8,698	99·3	
3. Experiment—						
Tokai	68	0	0·0	68	100·0	45
Robben Island ...	53	1	1·9	52	98·1	36
Experimental Station ...	191	69	36·1	122	63·9	5-40
	9,007	128	1·42	8,879	98·6	
Pretoria	1,400	8	0·57	1,392	99·42	
	10,407	136	1·31	10,271	98·693	

† " Experimental Investigation," &c., p. 42.

‡ The weights of these animals varied from 224 to 262 lbs., and the amount of serum per 600 lbs. live weight from 15 cc. to 35·0 cc.

slight symptoms, five had temperature reactions only, and eight had neither temperature or symptoms. There were no deaths. Of the two former nine proved to be immune from 73 to 244 days after the operation.

Captain Rogers is of opinion that the eight animals which did not react were endowed with a very weak immunity which would soon wear out. Formerly, I was of that opinion, but I now believe that the immunity so conferred is usually sufficient for all practical purposes.

Captain Rogers injected six of them with small doses of virulent blood and says that they reacted after this second inoculation. He seems to infer that this reaction was a slight attack of rinderpest, but I would point out that animals which have been immunised by fairly severe reactions with symptoms, also react after second injection of small doses of virulent blood.

The hill cattle were very much more susceptible to the disease; he writes that ". . . . good results could not be obtained by the unmodified serum simultaneous method in hill animals," and he advocates the use of large doses of serum simultaneously with virulent blood, to be followed by a second injection of 10 cc. of virulent blood eight to ten days later.

No doubt immunity can be conferred by this means at the expense of a waste of serum, but if Captain Rogers' experiments are carefully examined it is evident that he did not know the strength of his serum, that it must have been a remarkably weak sample, and that he was experimenting with doses which were far too small to be generally successful.

In fact, Captain Rogers in India has met, in a more marked form, a difficulty which we have also experienced in South Africa. Not knowing the differences which may exist between the animals from the plains and from the hills, because, unfortunately, I have never been to India, and it is quite possible that this difference may have its effect, there are facts which go far to explain the different ways in which these animals reacted after injection.

I gather that in the plains rinderpest is enzootic, and therefore animals living on the plains would be less susceptible to the disease, because they may have previously passed through an attack, or may have been born of mothers who, during the period of gestation, went through a mild attack. In that case they would be immune for a longer or shorter period. We noticed this with regard to calves born while their mothers were undergoing bile immunisation or shortly after. These little animals were almost invariably immune.

In the animals from the plains, then, it would not be surprising to find that the serum even when weak was successful, but any serum made from such animals would, in my experience, be weak.

I gather also that the disease is not so common in the hills, therefore animals born and living there would be less likely to have become immunised in the manner described above, and would therefore require larger doses of the serum to save them for a simultaneous inoculation with virulent blood. On the other hand, such animals would probably produce a strong serum if properly treated.

M. Nicolle has prepared serum by a method which he calls "La methode brutale." He injects simultaneously 25 cc. of serum and

large doses of virulent blood, even as much as 4,000 cc., and he found, as we had found at Kimberley, that animals so treated did not suffer more severely than those which had received very much smaller quantities of blood.

I have never experimented with such enormous doses as 4,000 cc., but I know from experiment that it is a matter of indifference, so far as the resulting fever is concerned, whether the first dose be 0·2 cc. or 200 cc.

He claims that in this manner much time is saved, as the animals are quickly ready for use. I do not wish to dispute this, but nevertheless the serum so prepared is much less powerful than that produced by the use of gradually increasing doses of blood.

Experiments in Egypt have proved this conclusively.

I do not wish to dogmatise, but I attribute the greater strength of the more slowly prepared serum to the greater number of reactions through which the animal has passed. Thus, there was generally not much difference as regards febrile reaction after the first fortifying injection of 10 cc. or the secondary dose of 100 cc., and the same may be said as regards the subsequent injections. It appears to me that it is this febrile reaction which increases the quantity of the immunising body in the serum, the more frequently the animal has reacted the better the serum.

I will now contrast the actions of the bile and of the serum.

I have already given my reasons for supposing that the bile in rinderpest is the habitat of a modified rinderpest organism. It also contains a small quantity of an immunising substance.

This substance, however, must differ considerably from the bulk of the immunising matter contained in the serum, because it acts very differently. The bile in a short time is capable of depriving a very considerable quantity of virulent blood of its infective power.

On the other hand, the serum, *in vitro*, has very little of such power. I found that when immunising serum was mixed with blood in small quantities, after twenty-four hours the mixture injected into a susceptible animal did not produce rinderpest, but that when the proportion of blood mixed with serum approached 1 per cent. the result varied, fever might or might not follow, in larger proportions the fever followed inevitably.

Again, as I have pointed out, if the blood and serum were injected simultaneously without being previously mixed the amount of virulent blood used was practically a matter of indifference.

It might be argued that the immunising serum contained two bodies, the one similar to that found in rinderpest bile, capable of killing the specific organism *in vitro*, and that this is in small quantity, and another which does not possess this power, but only acts through the intermediary of the living body.

If this should prove to be the case, then the blood must be capable of destroying an enormous quantity of the second substance *in vitro*, because after the mixture it no longer acts.

I have been careful not to use the word antitoxin, because I am not at all sure of the real nature of this immunising substance, which can only act through the intermediary of the cells or fluids of the living body.

The organism, too, which in the simultaneous method produces

permanent or at least prolonged immunity, is not the same as that to which bile owes its protective power, inasmuch as it is unmodified.

If an animal suffering from fever after the simultaneous inoculation be bled and a susceptible animal be inoculated with that blood it suffers from unmodified rinderpest; moreover, if an animal, immunised by the simultaneous method, be fortified by the use of a larger dose of virulent blood, the blood of that animal during the febrile reaction, injected into a susceptible animal, produces rinderpest.

The Kimberley simultaneous method is not without drawbacks in certain districts.

The serum itself is incapable of conveying disease; we may know that the animal from which the serum is prepared is suffering from redwater or *gal ziekte*, but that need give rise to no anxiety.

It is different with regard to the blood. If the beast from which the infecting blood is taken should happen to have been suffering from either of the diseases above mentioned, the result of the immunising inoculations may be very disappointing.

Blood used for this purpose at Kimberley was invariably carefully examined, and it was never taken from a beast which showed before injection great variations between the morning and evening temperatures. Nevertheless, more than once we experienced disappointment. Animals which had passed successfully through the immunising fever, and were in excellent condition, subsequently developed redwater, &c.

The quantity of blood examined was so small that when the organisms were few in number they escaped detection, but 1 cc., the dose usually employed, contained sufficient of them to be dangerous.

But this inconvenience can only be met with in districts in which those diseases are epizootic, and even then when the process is applied to indigenous cattle the loss is usually not great, because so many are already immune, but with respect to animals coming from areas free from these diseases the result may be very bad.

When one has to operate under circumstances which render a mixed infection probable or possible, the inoculation of blood should be given up and the French method of infection be used, *i.e.*, smearing the nose with fæces or mucus.

It is fortunate that the very formidable disease, Rhodesian bile fever, is not communicable by the injection of blood.

Unfortunately in the second epidemic of rinderpest in 1901, the difficulty experienced in providing animals on account of the war and the urgent necessity for serum, prevented me from making any experiments with a view of elucidating the many interesting questions which arise in connection with the production of immunity against rinderpest and immunity in general. The facts as regards rinderpest are not in my opinion in accord with those noticed with respect to the anti-diphtheritic serum. In fact, I am disposed to believe that each disease and each anti-serum must be studied separately, and that inferences drawn from one are only very roughly applicable to others. This is not surprising. The man who employed against measles the means found serviceable against scarlet fever would most certainly be disappointed.

One would like to be able to estimate the amount of good probably effected by the various precautions taken in the colony. This can only be imperfectly effected by comparing the loss actually suffered and

the loss which would have probably occurred had the disease been allowed to run riot.

The Cape Colony* is divided into seventy-seven districts; of these twenty = 26 per cent., escaped the visitation entirely.

Before the epidemic the number of cattle in the Colony was 1,639,435. The number dead of rinderpest amount to 575,864, therefore there remained 1,063,571 horned beasts.

But the twenty districts which were not invaded contained 147,883 cattle, therefore, those which were infected had 1,491,552 head, and as 575,864 died of rinderpest, the mortality was 38·61 per cent.†

In the districts of Mafeking when the disease appeared early and before preventive inoculation in any form was available, and where consequently only 0·097 and 0·666 per cent. respectively of the animals were inoculated, the loss was 97·29 and 92·76 per cent., or an average of 95·3 per cent.

Therefore it would be no exaggeration to assert that but for the preventive measure in one form or another, the average loss would be about 95·3 per cent.

If this supposition should be approximately correct, we may say that the result would have been :—

Before the epizootic	1,639,435	cattle.
Less 95·3 per cent.	1,562,382	,,
Remaining	77,053	,,
Probable loss	1,562,382	,,
Actual loss	575,864	,,
Probable saving	986,518	

These cattle taken all round before the onset of the disease would have cost not less than £10 per head. When people were selling oxen to the Rinderpest Station because they feared the loss of their entire herds the price paid averaged £6. We may therefore assume that the Cape Government by the steps it took saved between £6,000,000 and £10,000,000.

The Cape Returns are so compiled that it is impossible to gather from them the proportion which each of the several methods bore in this saving.

I know that the quantity of serum issued from the Kimberley station was 4,472 litres, which sold for £7 10s. per litre, the total value being £33,181.‡

* "Rinderpest Statistics for the Cape Colony, 1896-7-8," prepared by the Agricultural Department, Cape Colony.

† The publication above puts the mortality at 35·13 per cent., but no allowance is made for the twenty which were not invaded.

	Litres.		Value. £	s.	d.
‡For the Cape Govt.	2,073	=	15,542	10	0
,, British S.A. Co., Aug., Sept., and Oct., 1898	1,611	=	12,082	10	0
,, British S.A. Co., prior to Aug., 1898	646·4	=	4,848	0	0
Defibrinated blood	141·6	=	708	0	0
	4,472·0	=	£33,181	0	0

That amount was sufficient to inoculate 203,600 animals, and we heard of its use amongst 108,000; * we know also that the average loss was not more than 1·3 per cent.

Gentlemen, I have detained you an unconscionable time, my only excuse is that the subject is of great interest to myself personally, and I hope that the method here set forth may be of use elsewhere than in South Africa and Egypt.

Translations and Extracts.

IRREGULAR MOLAR TEETH IN THE HORSE.

BY BOURDELLE.

The patient was a seven-year-old mare. There were six well-developed molars on the right side, and the incisors were regular, with two small canines which were very close to the corners. The left lower molars were altogether absent, the edge of the gum being thin and sharp, without any trace of teeth. In the upper jaw there were six incisors and two canines which were regular and well developed, also six molars on the right side, the third one growing inwards. On the left side there were also six molars which were very sharp and bony, not being in the slightest degree worn down. The third superior molar on the left side was in reality formed of two portions which fitted exactly, but were separated by maceration. The free part consisted of a milk tooth which was fixed to the extremity of the permanent one, and the absence of the corner left molar allowed the upper ones to become greatly overgrown, and to interfere with mastication.

The case shows—

(1) That the accidental persistence of a temporary molar does not interfere with the development of the permanent ones.

(2) That diminution in number of teeth may give rise to serious consequences.

(*Revue Veterinaire.*)

DISINFECTION OF SKINS FROM ANIMALS WHICH HAVE DIED OF ANTHRAX.

BY PROFESSOR E. VON ESMARCH.

About 140 persons die every year in Prussia from anthrax contracted by handling the dead bodies, skin, &c., of animals, but very rarely through eating contaminated meat.

The author has studied the action of the following disinfectants: the action of the sun, carbolic acid, formaldehyde, corrosive sublimate,

* Much of the serum both in the Cape and Rhodesia was stored, and a considerable quantity went to Egypt, where it met with much success, in fact the Transvaal Government would have paid all its expenses if it had acted on my advice, and continued to manufacture serum for export.

and steam. When fresh skins are exposed to the sun on the internal surface they become sterilised in about seven hours if they do not contain any spores, and in thirteen hours if they do, but this plan is not reliable in practice.

If there are no spores they are sterilised by a 1 per 1,000 solution of carbolic acid in twenty-four hours, but are virulent for forty-eight hours after immersion in a 1 per 2,000 solution. In a 1 per 2,000 solution of formaldehyde skins are no longer dangerous after soaking for twenty-four hours.

In a 1 per 1,000 solution of corrosive sublimate and salt the skins, if free from spores, are sterilised in two hours, but the spores themselves are not destroyed under seven hours. Corrosive sublimate still retains its former reputation.

Steam should not exceed 70° C., and at this temperature will sterilise skins without spores in from five to ten minutes, and if medicated with formaldehyde in three minutes. Spores are destroyed in from four to eight minutes by steam medicated with formaldehyde in the proportion of 1 per cent.

(Festschrift zum Geburtstag von R. Koch.)

PRIMARY CANCEROUS GROWTHS OF THE SUPRARENAL CAPSULES IN THE OX.

BY M. HOLVOR HORNE, VETERINARY SECRETARY TO THE MINISTER OF AGRICULTURE, STATE VETERINARY LABORATORY, CHRISTIANIA.

THE author published an account in 1895 in the *Norsk Veterinar Tidsekrift* of certain tumours, probably of a cancerous nature, in the suprarenal capsules of the ox, which were reported on by Dr. Malm; other cases have been reported by M. Görig, director of the abattoir of Karlsruhe ("Primaris Corcinom der Nebenniere bei einer Kuh," *Deutsche tierarztliche Wochenschrift*, 1896, vol. iv., p. 305), and Sticker ("Uber den Krebs der Thiere," Berlin, 1902).

During the past ten years M. Horne has seen about twelve cases which he thinks are interesting from a pathological point of view. In most cases there is nothing abnormal to be seen in the living animal, but in one case of a sixteen-months-old ox there was great loss of condition, but the appetite remained good. In this case the tumour weighed about 15 lbs.

When the subject is in good condition and the growth is small, it may escape notice on *post-mortem* examination, or it may be buried in the peritoneal fat. In some cases the surrounding capsule is slightly œdematous, studded with patches of ecchymosis, and discoloured.

If a longitudinal section is made from before backwards the tumour is seen on the anterior border of the kidney, but separate from it, and there are minor adhesions. The renal substance is never implicated, although it is sometimes œdematous and ecchymosed. In some cases the kidney is indented by the tumour to a greater or lesser degree, depending on the size of the growth, and in this region it is atrophied.

In some cases the growths implicate both suprarenal capsules, in others only one, and vary in size up to 16 lbs. They are usually

spherical, with a smooth surface, and covered with a fibrinous membrane, and are of a soft consistency. On section they are seen to be of a variable colour, studded over with numerous hæmorrhagic patches, and in some cases there may be cystic cavities, containing a sero-sanguineous fluid. In some cases the author has found that the tumours have undergone caseous degeneration and calcareous infiltration where they assume a darker yellow tint.

The structure of the tumours are encephaloid carcinomata, the cellular veins being separated from each other by a stroma of connective tissue, in which the cells are scattered in an irregular fashion. At certain points there are small lobules formed out of cells resembling those of the cuticular substance of the capsules, round or polyhedral, with bright distinct nuclei; in others, chains of epithelial cells of a cylindrical form, resembling those of the medullary substance of the capsule. In the connective stroma there is a network of capillary vessels that often give rise to profuse hæmorrhage, and in one of the author's cases the growth had caused ulceration of the vena cava for several centimetres.

(*Revue Generale de Médecin Vétérinaire.*)

TUBERCULOSIS OF THE ELBOW-JOINT IN THE OX.

BY DR. WYSSMANN (NEUENEGG).

Hess and Gullebeau (*Schweizer Archiv*) have reported numerous cases of tuberculosis localised in the articulations, the sheaths of the tendon and the bursæ. Utz (*Bad tierarztl Milteil*, 1881) also mentions the same. On the contrary, M. Strabel ("La Rheumatisme Articulaire chez la Bæuf," *Revue Generale*, June 1, 1903, vol. i., p. 629) considers tuberculosis in this region as being of rare occurrence, and that such cases are rheumatoid.

Tuberculosis in order of frequency attacks the femoro-tibial articulation—the knee, back, elbow, and fetlock. Hess mentions a case of its localisation in the elbow. The first was in a young bull 15 months old that had been lame for five weeks, and in which the lungs and liver were found to be diseased when slaughtered. The second was a seven-year-old cow that had eczema of the neck, and was destroyed six days after the commencement of the lameness. Lesions were found in both the mesenteric glands and the lungs. The author mentions another case in a two-year-old cow that had been lame for three weeks. There was great difficulty in standing, and the skin was thickened and very painful. Movement of the joint did not cause crepitation, the olecranon muscles were atrophied, and there was a characteristic cough with râles on auscultation. The animal was slaughtered four weeks after, when the lungs and liver were found to contain tubercular patches that were undergoing carious or calcareous degeneration. In the sternum there was a carious patch the size of an apple. The articular cartilage on the lower extremity of the humerus was of a purple colour and deeply abraded. A soft pale pink tumour, which contained several collections of yellow carious material the size of the tip of the finger, occupied the posterior portion.

(*Wochenschrift für Tierhaelkunde.*)

THE PRESENCE OF SUGAR IN THE URINE IN THE DOMESTIC ANIMALS.

BY M. CH. PORCHER, LYONS VETERINARY SCHOOL.

THE presence of sugar in the urine is not much counted upon for diagnostic purposes in veterinary medicine, which may perhaps be accounted for by the difficulty there is in obtaining it: but nevertheless, some valuable aids in the elucidation of the problems of disease are lost through the neglect of making a systematic examination, and it is to be hoped that it will be more frequently had recourse to than has been the custom.

Lavigne, speaking of diabetes in man, says: "There is no true description of diabetes in the earlier stages, although there are many excellent ones of it when confirmed." This also holds good with animals, and many valuable symptoms are not recorded, either because the patient is seen too late or because they are not thought about. No doubt the testing of the urine requires a certain amount of care and skill, and frequently the results are negative; but considering the valuable indications that are given, it should be had recourse to more frequently than is the custom.

The different forms of sugar found in human urine are glucose, levulose, galactose, lactose, saccharose, maltose, tromaltose, and pentose; but some of these are exceedingly rare, and have never been isolated in the pure state, such as levulose, tromaltose, and maltose, and in veterinary medicine the only ones that have been detected with any certainty are glucose, lactose, and galactose. When there is any appreciable quantity of sugar in the urine, it is accompanied with polyuria. This is caused by an excess of sugar in the blood, generally in the form of glucose, although lactose is sometimes found in cases of milk fever. The polyuria is the result of the excessive amount of sugar in the blood, and this in its turn is due to polydyspepsia, and may be explained by the fact that, in order to be eliminated, sugar requires to be greatly diluted.

The quantity of the urine is increased; it is pale in colour, and has an aromatic smell, sometimes that of acetone; the density is increased, also the amount of urea and chlorides. The amount of sugar is difficult to estimate, as but few observations have been made; but Saint Cyr records a case in a dog where 83·46 grammes were detected. However, when the urine is pale, of a heavy specific gravity, and large in quantity, diabetes is to be suspected.

A golden rule that should be remembered is that if sugar is detected in the urine, the test should be repeated, as a single one is valueless, and the examination should be repeated for several days running.

There is a great difference in glucosuria and diabetes. In glucosuria the quantity of sugar is small and is not constant, whereas in diabetes, on the contrary, the quantity is large and constant. Glucosuric diabetes is more persistent, also the symptoms.

Glucosuria.—The conditions under which glucosuria is seen may be divided into four heads: A, Experimental; B, Therapeutic and Toxic; C, Physiological; D, Pathological.

A. *Experimental.*—This heading can be subdivided into (1) alimentary, (2) phloridsic, (3) from puncture of the floor of the fourth ventricle of the brain.

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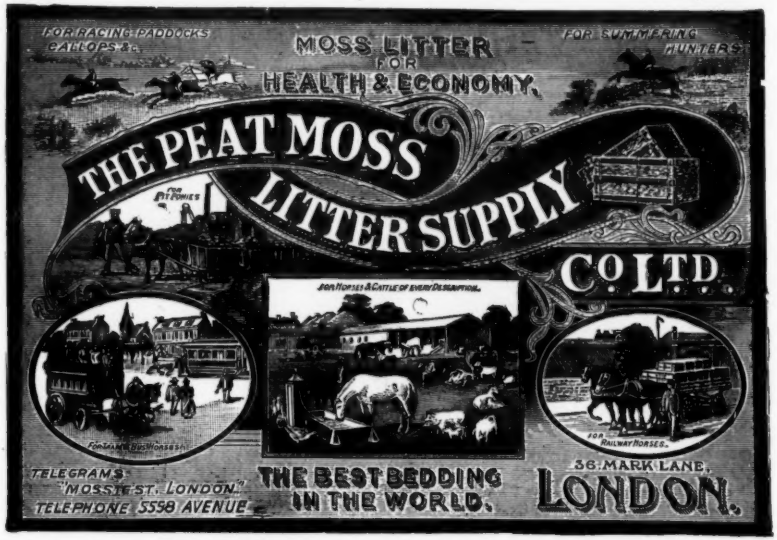
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The "COUNTY GENTLEMAN," *April* 1, says: "Chief among the merits of Peat Moss are the ease with which it is stored; the small danger it offers as regards fire compared with Straw; non-conductivity; the great powers of absorption; its antiseptic properties, and the ease and advantage with which it can be used as Manure in town gardens and in the country."

(1) *Alimentary.*—This was recorded in the dog by Budge in 1844, and the cat by Schmidt in 1850, after feeding on large quantities of sugar. From a clinical point of view, it is useful to determine the condition of the liver, whether it has a certain amount of permeability under certain conditions, or when taken fasting it points to hepatic insufficiency in the formation of glycogen.

(2) *Phloridsic.*—Phloridzin is a glucoside which, when injected subcutaneously, rapidly gives rise to a special form of glucosuria from excessive permeability of the kidney. The test by phloridzin is one that is used to diagnose renal insufficiency.

(3) *Glucosuria* from puncture of the fourth ventricle of the brain is evidently experimental, but it is not too much to say that the mechanism by which it is caused is the same that brings it about in certain pathological conditions, such as milk fever and rabies.

B. *Therapeutic and Toxic.*—After the absorption of certain drugs such as antipyrin, sulphonal, turpentine, &c., and as symptoms of certain poisons, the urine rapidly acquires the property of reacting to Fehling's test. Usually this is due to glucose, but since the glycuronic compounds have been better understood there is no doubt that certain cases of therapeutic and toxic glucosuria are due to the presence of them in the urine, and not to glucose.

(*a*) *Medicinal.*—It is well to remember that certain drugs endow the urine with properties of reduction, as if it really contained sugar, the chief being antipyrin, chloral, sulphonal, chloroform, turpentine, antifebrin, rhubarb and senna.

(*b*) *Toxic.*—This class can be divided into two.

(1) Glucosuria without any appreciable hyperglycæmia. The drugs that give rise to these conditions to a certain extent act like phloridzin, altering the permeability of the kidney; in this condition there is usually also albuminuria. Amongst the poisons that induce this condition are cantharides, chromic acid, arsenic, phosphorus, mercury, when given in poisonous quantities, and chlorate of potash.

(2) Glucosuria with chronic hyperglycæmia is usually the result of subacute intoxication. Prolonged asphyxia and poisoning by carbolic acid are usually accompanied by glucosuria. Possibly it is caused in the same way as the glucosuria of anæsthesia.

Subacute poisoning by curare, chloral, strychnine, morphine, atropine, and nitrate of amyl is often accompanied with glucosuria.

C. *Physiological.*—Under this heading comes the glucosuria of pregnant women, and in the domestic animals when the quantity of sugar per litre amounts to 12 or 15 grammes.

As has been previously explained, the mammary gland forms lactose as well as glucose, and the last is diverted into the circulation in excess of the normal quantity being ultimately formed into lactose. This excess is thrown into the circulation by the liver, which insures the constant formation of lactose.

Although both these physiological acts form sugar of milk with excessive production of glucose that is ultimately formed into lactose, they are to a certain extent dependent on each other, they are not simultaneous, and the super-production of lactose precedes the full functional activity of the gland, and it not being ready to form it into lactose it is eliminated by the kidney and gives rise to glucosuria.

This condition is not to be confounded with true diabetes, as there

is neither polyuria or polydyspepsia. It is fairly constant, but not constant in degree or in the true form, and disappears with parturition, lactose being found in its place.

In animals such as the cow and goat, where the milking function is highly developed, the glucosuria that is present in women is not found. In the domestic animals the mammary gland is active before parturition, and lactosuria not glucosuria exists.

D. *Pathological.*

(1) *Nervous Glycosuria.*—The etiology of this form has had considerable light thrown on it by the famous experiment of C. Bernard, of pricking the floor of the fourth ventricle, and as a general rule it may be stated that all diseases of the brain and cord will cause glucosuria when this part is attacked. This is seen when tumours gradually exercise pressure on the glycosuric centre. In the case of a man suffering from typical diabetes, who died in a state of coma, Ivan Michael found a cyst adhering to the floor of the fourth ventricle. It would be interesting to study the condition of the urine in sheep affected with cœnurosis.

In the human being glucosuria has been noticed in cerebral apoplexy, tumours in the brain, cerebro-spinal meningitis, general paralysis, and tabes.

The glucosuria, which is not uncommon with chorea, as well as other nervous diseases in man, has been noticed in the dog suffering from chorea by Nicolas and Rabieaux (*Journal de Physiologie et de Pathologie Generale*, 1902, p. 95), and Roger (*Revue Vétérinaire*, December 1, 1904, p. 813) has recently recorded one in a stallion with dourine.

The diagnostic importance of glucosuria in rabies has been recorded by Colasanti ("Glyco-albuminuria rabbica," *Boll. R. Accad. Med. di Roma*, vol. xix., 1894), Porcher (*Bulletin de la Soc. Central Méd. Vét.*, 1898), and Nicolas and Rabieaux (*Journal de Physiologie et de Pathologie Generale*, 1902, p. 95.)

Regarding its value, Cadeac ("Semioligie et Diagnostic") says: "The symptom (*glycosuria*) which is seen in a number of complaints has not a very great value for diagnostic purposes." In this the author appears to be mistaken, as it is valuable evidence of rabies, especially in doubtful cases, and in doubtful cases the tests should always be carried out, especially in suspicious cases which have died with nervous troubles.

It is necessary to point out the difference between rabic glucosuria in the carnivora and herbivora. In the dog the presence of glycosuria is looked upon as corroborative of the appearances seen in the dead body. It is not always present, and may come on late in the case; its absence, however, must not be looked upon as proving the non-existence of rabies.

In the herbivora the presence of glycosuria is of real diagnostic value, although it is seen in other diseases such as milk fever. It is of value not only on account of its constancy, but also because the symptoms in this class of animal are obscure.

As a rule, rabic glucosuria is more intense in the herbivora than the carnivora.

In milk fever there may be either glucosuria or lactosuria.

(2) *Infectious Glucosuria.*—Nocard and Motas (*Annales de l'Institut*

Pasteur, 1902, p. 257), and Maignon (*Société des Sciences Veterinaires de Lyon*, 1903, p. 287), record the presence of glucose in canine piroplasmosis, varying from a few decigrammes to 6 grammes per litre; and M. Mouquet (*Recueil de Médecine Vétérinaire*, 1893, p. 412) found 10 grammes of glucose to the litre in a case of hæmoglobinuria. The glycogenic function of the liver is interfered with, and M. Mouquet has twice seen horses suffering from glucosuria (*Recueil de Médecine Vétérinaire*, April 15, 1894). In the first case, in which there was enteritis and degeneration of the hepatic cells, he found 15·40 grammes of glucose per litre, and in the second one 5·50 grammes.

Diabetes.—The cases of diabetes in the domestic animals that have been recorded are few, and these must be accepted with caution. Although the etiology of diabetes is not well understood, it can be divided into two types—hepatic and pancreatic.

According to Thurnesse, Frohner, Saint Cyr and others, diabetes is hepatic in the dog, but Liénaux mentions a pancreatic one, the gland being entirely atrophied.

The cases of diabetes that have been recorded in the horse and ox are not very reliable, and it is only the polyuria that has attracted attention. Cadéac, in stating that diabetes is a symptom of congestion of the cord, and Mouquet of hepatic affections, have confounded the terms diabetes and glucosuria. In diabetes the quantity of sugar in the urine is generally high, Peteaux and Saint Cyr have found as much as 83·50 and the author 48·30 grammes in the dog.

Lactose.—Lactosuria is only seen in milking animals, lactose being a sugar formed by the mammary gland. If sugar is found in the urine of a milking animal, its nature must be ascertained. If it is lactose it is usually only a natural production, but if glucose it is altogether a different matter.

There is always lactose in the urine of pregnant cows at th moment of parturition, sometimes as much as 13 grammes and over to the litre (Porcher and Leblanc, *Société Centrale de Médecine Vétérinaire, see* July 1902, p. 661). If for any reason the cow is not milked or is not milked clean, the lactose is reabsorbed and partly eliminated in the urine. Bitches sometimes give milk when they are not pregnant, and in such cases there is constantly sugar in the urine, as the lactose is reabsorbed almost as quickly as it is secreted.

Milk Fever. (*a*) *Glucosuria.*—It would appear to be analogous to that caused by puncture of the floor of the fourth ventricle. It comes on rapidly, lasts but a short time, and disappears quickly. It is, no doubt, due to the gland not being able to cope with sufficient rapidity with the quantity of glucose that is formed.

(*b*) *Lactosuria.*—Lactosuria is also of bulbar origin, but, the gland being active, the glucose is formed into lactose, which is eliminated by the kidney.

There is always sugar present in the urine in case of milk fever, but as has been shown, it varies in both quantity and quality. In the human urine there are other sugar compounds besides glucose and lactose, but in veterinary medicine they need not be taken into consideration.

(*Revue Générale de Médecine Vétérinaire.*)

AVIAN DIPHTHERIA IN THE ISLAND OF CUBA.

BY DR. JUAN DAVALOS (HAVANA).

THE disease has been known in Cuba from time immemorial under the name of "moquillo," and causes great loss in poultry annually. Bacteriological examination has demonstrated a cocco bacillus identical with that which Guerin discovered in 1901, and in all its characteristics, except its mobility, it resembles the Pasteurella of Lignières, whose movements continue after its destruction by heat or antiseptics.

The disease exists in both an acute and chronic form, the former killing in from five to fifteen days, and the latter lasting for several months.

The acute form commences with dulness, loss of appetite and anorexia; the bird being dull, the wings drooped, the feathers standing on end, and the respirations quickened and difficult. The membrane of the mouth is coated with a viscid fluid with small projecting patches, consisting of a yellow-coloured, caseous exudate, which is only slightly adherent, but which is extended over the pharynx and larynx. Sometimes the lesions extend to the nasal cavities. and it is not uncommon to find a specific conjunctivitis with or without ulceration of the cornea. There is asphyxia, diarrhœa, and anorexia, and death takes place in a state of collapse.

In the chronic form only the local lesions are apparent, and there is a caseous exudation spread over the mucous membrane of the mouth, especially on the tongue. The bird refuses food, and the lesions spread to the eyes and nostrils. Swallowing and respiration become painful and difficult; diarrhœa, with extreme emaciation and anæmia, sets in, followed by death in a short space of time. Pigeons are extremely sensitive; four or five times the fatal dose for these birds is harmless to the fowl.

The microbe is always to be found in the membranes. There appears to be no connection between this form and human diphtheria.

(*Revue Générale de Médicine Vétérinaire.*)

EMBOLISM OF THE PULMONARY ARTERY.

BY DR. HORET KAPPEL (LEIPZIG).

IN the summer of 1901 the author was called in to examine the body of a horse which had been slaughtered, and which during the night had suffered from a severe attack of dyspnœa. At first sight all that could be discovered was valvular insufficiency and slight emphysema at the edge of the lung, but on further examination a greyish-red clot, 10 to 12 centimetres long, was found in one of the larger branches of the pulmonary artery at the lower third of the lung, and no doubt exists in the mind of the author that this was the cause of the trouble. As the result of this case Dr. Horet Kappel made a systematic enquiry into the matter on which, with the exception of a short note by Kitt (*Pathol. Anat. der Hassäwgethiere*, 1901, p. 188), veterinary literature is silent.

The author recommends that an incision be made over the lower

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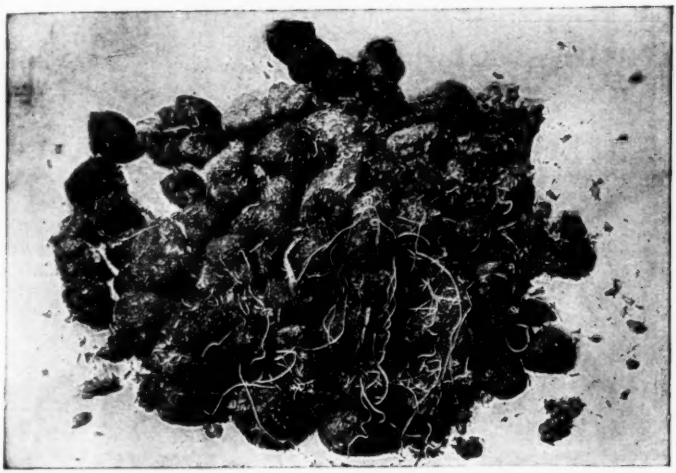

Photograph of a portion of the manure from "IRENE" before being fed with Molassine Meal.

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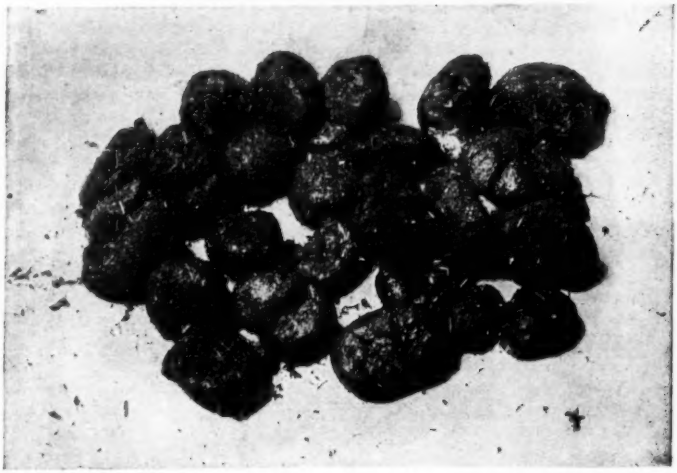

Photograph of Manure from "IRENE" after feeding with Molassine Meal.

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third of the principal tubes of both lungs, and a second one parallel to the first, 10 to 11 centimetres lower down, and the canal exposed by dissection. Out of 1,977 lungs, 29, or 1·47 per cent., were found to have arterial obstructions. This is small compared to the 8 to 10 that Virchow found in the human subject, and his suspicions were excited that the plan he adopted was ineffectual. He therefore carefully dissected out the pulmonary artery down to its minute branches, and in 100 cases found traces of obstruction in 9, which gives the same percentage as in the human being.

In the case noted by the author the obstruction varied according to the age of the lesions. Recent clots were more or less adherent to the walls of the artery. The largest measured 2½ centimetres by 28 in length, more of an elastic consistency and soft in the centre. In the older clots the tissue was invaded by granulations extending from the periphery, and after a time the only trace left were fibres of a brown or greyish-white colour. Out of 38 cases that came under the author's notice the clots were completely organised in 3, in process of organisation in 7, and in various conditions in 10.

The formation of the embolism in different parts is due in the first place to the direction of, and in the second to the diameter of, the artery, and generally in the posterior third of the large lobe. Out of the 38 cases reported, in one case only the obstruction existed in the anterior and twice in the middle lobe.

The origin of the embolism is a matter of dispute, Virchow (1856) maintaining that it was venous, and Oeth (1900) that it was cardiac. The author found that out of 38 cases of pulmonary embolism 24, or 63 per cent., also had venous thrombosis. The frequency of this is well known, and Kitt points out that they are most frequently found in the veins of the sheath, scrotum, pituitary membrane, and skin.

All diseases of the veins accompanied with thrombosis, such as endothelial degeneration, wounds, dilatation, and inflammation, predispose to pulmonary emphysema, and the process is favoured by age, anæmia, cachexia, &c. Certain diseases also are active factors, viz., anasarca, erysipelas, infectious pneumonia, strangles, &c. Venous thrombosis is also common in enlargement of the extremities and in the neighbourhood of diseased joints.

The symptoms are not very well marked, but, as the author points out, a number of conditions the origin of which at present are obscure can be traced to pulmonary thrombosis.

The symptoms consist of shortness of breath, coming on during work, giving rise to dyspnœa that may be so bad that the animal falls down. When at rest there is quickened respiration, with marasmus, loss of appetite, and incapacity for work. In most cases the respiratory troubles improve in two or three months, as the clot becomes organised, but recurrence is always probable from the formation of fresh emboli. Examination of the chest both by auscultation and percussion gives no indications.

The author arrives at the following conclusions :—

(1) That about 9 per cent. of horses suffer from embolism of the pulmonary artery.

(2) That out of these 63 per cent. suffer from thrombosis of the veins in the hind-limbs.

(3) Cases generally are seen in old horses.

(4) In most cases there are but few symptoms visible during life except disorganised respiration and a peculiar cough; death may take place from asphyxia caused by arrest of the circulation in the pulmonary artery.

(5) As a rule, circulation is returned in the obstructed vessels by the clots contracting and allowing of the passage of the blood.

(*Zeitschrift für Thiermedicin.*)

GLANDULAR TUBERCULOSIS IN SLAUGHTERED ANIMALS.

BY STROH.

THE author's observations were made in the Abattoir of Augsbourg from 1902 to 1904:—

	Tubercular Animals.	Cases of Tuberbular Glands.	Percentage.
Oxen...	1,254	18	1·43
Bulls...	1,101	22	1·99
Cows...	4,549	137	3·01
Calves	310	71	22·90
Pigs ...	1,425	205	15·78

From these batches it would seem that the muscular glands are more frequently attacked in adult animals than in calves. The percentage of the different glands is as follows:—

	Prepectoral		Axillary		Crural		Popliteal		Ischiatic	
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Oxen	12	37·50	4	12·50	6	18·76	5	15·62	5	15·62
Bulls	11	33·33	2	6·06	6	18·19	9	27·27	5	15·15
Cows and heifers	50	24·03	9	4·32	52	25	60	28·85	37	17·80
Calves	53	40·78	9	6·93	29	22·30	31	23·05	8	6·14
Pigs	142	52·59	—	—	92	34·08	36	13·33	—	—

It would appear therefore that in adult bovines the prepectoral and popliteal glands are most frequently attacked, and in pigs the prepectoral. The superficial vaginal glands in cows and sows are often affected. In 1902-4 Stroh found them invaded in 4 oxen, 97 cows, 2 calves and 178 pigs. The mammary gland was tuberculous in 1·74 per cent.; in 51 cases there were complications of the corresponding glands, and in 82 cases the glands alone were affected.

(*Zeitschrift für Fleisch und Milchhygiene.*)

THE ELIMINATION OF DRUGS IN THE MILK.

BY VAN (ITALLIE).

In a series of experiments made in conjunction with M. Senshoek, the author has made the following observations on the elimination of medicine given to animals in the milk.

He remarks on some experiments by Stumpf, who found that iodide of potassium decreased the secretion if given for a short period, but if prolonged increased it, that alcohol-morphine and lead caused no alteration, while salicylic acid increased it.

Stumpf and Winternitz's experiments were made on goats, and the author's on cows. His investigations were conducted with the alkaloids, iodide of potassium, salol, salicylic acid and turpentine.

Sulphate of physostigmine, 0·05 gramme, and hydrochlorate of pilocarpine, 0·25 gramme, given subcutaneously, could not be detected in the milk by Stass and Otto's or the physiological method.

Opium in 8 gramme doses daily for four days had no influence on the secretion, and no traces were present in the milk.

Iodide of potassium in 5 gramme doses daily for four consecutive days could not be discovered in the milk. Stumpf, however, with Pitthausen's method (sulphate of copper and potash, with the addition of sulphuric and nitric acids), demonstrated the presence of 0·000·006 to 0·000·058 per cent. of iodine in the milk. He used 25 cc. of milk diluted with four times its volume of water.

Van obtained a slight reaction. The milk was treated with soda evaporated and calcined, the carbon that remained being treated with warm water. One part of the solution was concentrated by evaporation acidified by dilute sulphuric and treated with fuming nitric acid. The chloroform test was then used to detect the iodine that had been set free, when a clear red colour was given denoting about 1-1,000 milligramme of free iodine. The author thinks that in practice iodide of potassium need not be considered.

Salicylate of soda in 20 gramme doses daily for four days was not detected. The milk was treated with ether evaporated at a low temperature, and the residue treated with warm water filtered, and the filtrate treated with perchloride of iron, but there was no violent reaction given. A second portion of the milk was rendered alkaline and treated with ether, and then with dilute sulphuric acid till coagulated, when it was heated to 70° C. and filtered. When cooled the filtrate was treated with ether and then tested for salicylic acid.

Salol was given to a cow in 5 gramme doses for six days, and the result were negative.

In 1879 Pauli noticed salicylic acid in the milk of ninety calved cows that had been given salicylate of soda, but Stumpf could not detect it under the same conditions in that of goats.

Turpentine.—Turpentine cannot be recognised; and in summing up his work, the author arrives at the conclusion that only iodide of potassium leaves any trace of its presence in the milk.

(*Tijdschift voor Veeartsenijkunde.*)

PERFORATION OF THE SMALL INTESTINE BY ASCARIDES.

BY M. BARTHELEMY.

THE patient, an entire barb, 9 years old, was attacked with colic at 2 p.m. on June 4, 1903. He had not been worked that morning, and at 9 a.m. had eaten his usual feed. At one o'clock he became unwell. There was no previous history of colic. The symptoms were all the usual ones, with tympanitis on both sides; pressure in the flank caused pain. The diagnosis was indigestion, and the prognosis was bad. Treatment consisted of stimulating liniment to the abdomen, with hypodermic injection of 15 centigrammes of pilocarpine, ether, and caffeiné. A litre of coffee and alcohol was given in a draught. At 7 p.m. mustard was applied to the abdomen, but without any result; the patient died at 8.15 the same evening.

Post Mortem.—The peritoneum was greatly congested, and contained about three litres of blood-stained serum. About twenty ascarides were found in the anterior portion of the small intestine, in which was an ovoid mass about 10 by 6 ins, situated about 2 ft. 10 ins. from the pylorus. It consisted of food and blood clots that were more or less adherent to the intestine, and in the middle of which was an ascaris. The mass was contained between two folds of the peritoneum, which were of a deep red colour, and at the place where the parasite was fixed about $\frac{1}{16}$ of an inch thick, so as to form a pocket. In the intestinal wall was a circular opening $\frac{3}{8}$ of an inch in diameter, through which the parasite and the food had evidently escaped. The edges of this opening were slightly infiltrated, but the mucous and muscular coats were not very much separated from each other, and clearly it was *ante-mortem* and caused by the parasite, the latter giving rise to the peritonitis. The opening was a clean perforation, and not a rupture.

(*Bulletin de la Société Centrale de Médecine Vétérinaire.*)

TUBERCULAR OSTEITIS IN A HEIFER.

BY M. MOUSSU (ALFORT).

TUBERCULAR osteitis in cattle is exceedingly rare compared to other forms of the disease, and this case is only the second that the author has met with in ten years. M. Hamoir has recently given a good description of the vertebral form, and M. Lienaux has reported one in the upper jaw.

The case reported by M. Moussu was a Dutch heifer, 18 months old, which was brought to the Alfort College for treatment on April 3, 1905, for an injury to the left temporal region. About a month previously she had suffered from suppurative otitis of the left ear, of a very obstinate nature, with a diffused swelling around the base, this being hard, painless and non-inflammatory.

The appetite had diminished, but apparently this was more from difficulty in moving the temporo-maxillary articulation than from any morbid condition of the digestive apparatus. All the other functions were normal, and the lesion appeared to be purely local. From his experience of a similar case, the author used the tuberculin test, but without any result, and the supposition of a dentigerous tumour was dismissed, as there was not the peculiar hardness or sharp line of delimitation characteristic of these growths. Upon exploratory puncture being made with a small trocar, the point came in contact with the bone at a very short distance below the skin, and penetrated into it, showing that it was considerably softened, and the diagnosis was doubtful.

The animal got rapidly worse, and the left articulation could only be moved imperfectly, while the left temporal form was gradually filled up. The case appeared to be hopeless, and the animal was destroyed.

On *post-mortem* examination an enlargement the size of two clenched fists was found to be implicating the whole of the left side of the cranium, extending from the upper part of the temporal fossa to below the temporo-maxillary articulation, and embracing the external auditory canal.

The mass was irregularly hemispherical in shape, and adherent to the outer table of bone. The disease commenced in the cancellated tissue spreading externally to the compact, which was perforated in several places, but the connective tissue and skin were healthy.

On section the growth was found to consist of a bony framework enclosing numerous tubercular masses, which were hard and of a characteristic yellow appearance. The outer layer of bone was thicker than the septa, and was perforated in several places, allowing the passage of prolongations from the interior mass.

The retro-pharyngeal glands were invaded, but not the bronchial or the lungs. The case is curious on account of its rarity, also because the diagnosis of such is difficult, and in this one the use of tuberculin failed to give the usual reaction.

(*Bulletin de la Société Centrale de Médecine Vétérinaire.*)

THE NUMBER AND VALUE OF HORSES IN THE UNITED STATES.

THE number of horses, according to the Department of Agriculture returns, has risen from 14,213,000 in 1890 to 17,057,000 in 1904. In the same time their value has risen from 978,000,000 dols. to 1,200,000,000 dols. The greatest scarcity was in 1900, when but 13,597,000 were found. All this in spite of the fact that fifteen years ago it was said that the cable, the trolley and the bicycle—and later the auto-trucks and automobiles—were destined to put the horse out of business.

(*American Veterinary Review.*)

PIROPLASMOSIS IN HOLLAND.

BY A. DE JONG.

THE dourine may be diagnosed in the living animal, but usually it is only done on *post-mortem* examination, and thus it is not very satisfactory. With blood that has been collected for any length of time the organisms are ill-defined, and are more or less circular and small, whereas in fresh blood they are larger, numerous, and distinctly pyriform.

The author has already published two cases of bovine piroplasmosis in Holland (*Tijdschrift voor Veeartsenijkunde*, vol. xxix., p. 531, and vol. xxx., p. 431; also *Gasille op Noordwelle*, in 1904).

Numerous piroplasms in the red corpuscules were found in blood from the liver, kidneys, and spleen. The parasites generally were small, one, two, and even more being found in the same globule. They were round and pyriform, but not as large as the figures given by the author in 1902-1903, or those by Wassenaar, of Steenwijk.

In a case reported by Overbeek, of Steenwijk, they were found in the blood of an ox infected with ticks (*Ixodes redivivius*). At Midwolde, hæmoglobinuria is well known, and most of the cattle in the neighbourhood have ticks on them. Old ones die, but those that recover are unsound.

Piroplasmosis does not always cause hæmoglobinuria, and the author mentions the following fact. An animal, supposed to be suffering from charbon, was slaughtered at Leyde in 1903, and on *post-mortem* examination fifteen litres of blood were found in the abdomen. There were no symptoms of charbon, but the various organs and glands were enlarged and hæmorrhagic, especially the liver and spleen, and the conjunctiva was a deep yellow. There were piroplasma present in the blood in small quantities, but very distinct, chiefly found in the blood from the spleen.

The author has received from the district of Warmond some interesting facts relating to a dourine in oxen in which there are lesions of the spleen, liver, and kidneys, but in which there were no symptoms of hæmoglobinuria. De Jong thinks that it is due to piroplasmosis, as he found, "although with difficulty," some hæmatozoa in the blood; also, in 1903, he noticed the same in a fresh outbreak at Katwijk.

(*Tijdschrift voor Veeartsenijkunde*, March, 1904, p. 256.)

CERVICAL ACTINOMYCOSIS.

AT a meeting of the Society of Dermatology and Syphilography of Paris, held on January 11, M. Pautrier showed a patient who was an agriculturist, and who had been in the habit of chewing ears of grasses. In September, 1903, he presented a tumour at the angle of the jaw on the left side, in which were found the characteristic yellow granules of actinomycosis. To remove it a portion of the angle of the jaw had to be taken away as well. Four months later cutaneous lesions began to develop in the neck, and here also were found actinomycotic granules. Treatment by iodides had not so far brought about any improvement. —*Dental Surgeon.*

Reviews.

ANIMAL DENTISTRY AND DISEASES OF THE MOUTH. By L. A. MERRILAT, V.S. 261 pages, 165 illustrations. Price 12s. net. Published by Baillière, Tindall and Cox, London.

This volume by Mr. Merrilat, Professor of Veterinary Surgery in Chicago Veterinary College, and late Professor of Dentistry in the McKillip Veterinary College, is the first of three volumes on the subject of veterinary surgery, and is well worth a place on the bookshelf of every veterinary practitioner in England and America.

The subject of animal dentistry is one which has not, until recent years, received as much notice as it should have done, and in America it is one to which very great attention is now paid. There is no doubt that many ailments from which all classes of animals suffer can be traced to diseases of the teeth, and Professor Merrilat has illustrated a large number of abnormalities and maladies from which the teeth may suffer, together with the most up-to-date methods and instruments utilised for their prevention and cure. We notice amongst the illustrations that on page 237 Cherry's operation, which is so well known in England as of value for the closure of wounds over which the skin cannot be made to meet (and be permanently retained in position) on account of tension put upon it, is clearly illustrated, although it is claimed as being something new and as being the original idea of the author's brother, Professor Edward Merrilat. It is said that there is nothing new under the sun, and in England at all events, this operation has always been spoken of as "Cherry's operation" for the last forty years or more, and is supposed to have been an original idea on the part of a gentleman of this name, who was once a veterinary surgeon in the British army.

With the exception that this operation ought not to be claimed by Mr. Edward Merrilat as being original, and another figure of a dog's speculum on page 174, which is termed "White's," but which originally was devised and fully described in the English veterinary periodicals some years ago by Professor Hobday, of the London Veterinary College, we have nothing but praise for the arrangement of the book. Some of the descriptions might be more lengthy and given in a little more detail, but the whole is interesting reading. The part of the publisher has been done well, and the book is neatly bound and got up.

SPECIAL REPORT ON THE DISEASE OF CATTLE. By numerous Veterinary Authorities. Issued by the United States Department of Agriculture, Bureau of Animal Industry.

In America it is the custom for the Department of Agriculture to issue every year in volume form a number of valuable notes on the diseases of the domesticated animals, the reports being written by thoroughly up-to-date and competent veterinary surgeons, each of whom are specialists in the particular branch which they take up.

As stated in the preface, they are prepared for the use of the farmer and stock-owner rather than the student or veterinarian, but upon

perusal it is found that there are very many pages which the qualified veterinary surgeon can read with pleasure and profit. The illustrations are exceedingly good, many of them being coloured, and the subjects chosen are of every-day interest.

The volume is only to be obtained by the special favour of the Bureau of Animal Industry, and is distributed without charge.

Correspondence.

CANINE SURGERY REVIEW: A CORRECTION.

DEAR SIR,—In the review of "The Surgical Diseases of the Dog and Cat" (Hobday) in your last issue, one paragraph reads as follows: "In our hands the use of 30 m. of 40 per cent. solution of cocaine has given far more satisfactory results by hypodermic injection than 60 m. of 2 per cent."

It should have read "30 minims of 4 per cent. solution" (not 40 per cent.).

Trusting that you will allow this to be corrected.

Yours faithfully,

G. H. W.

Letters and Communications, &c.

Capt. Nicholas; Colonel Nunn; Mr. Pollock; Mr. Brown; Dr. Turner; Mr. Slipper; Mr. Bond; Mr. Linton; Prof. Bradley.

Books and Periodicals, &c., Received.

Diseases of the Horse's Foot (Reeks); The Creamery Journal; The American Veterinary Review; Transvaal Agricultural Journal; Journal of the Royal Army Medical Corps; Department of Agriculture (Ireland); Board of Agriculture; Clinica Vétérinaria; Cape Agricultural Journal.

NOTE.—All communications should be addressed to 8, Henrietta Street, Covent Garden, London, W.C. Telephone, 4646 Gerrard. Telegrams, "Baillière, London."

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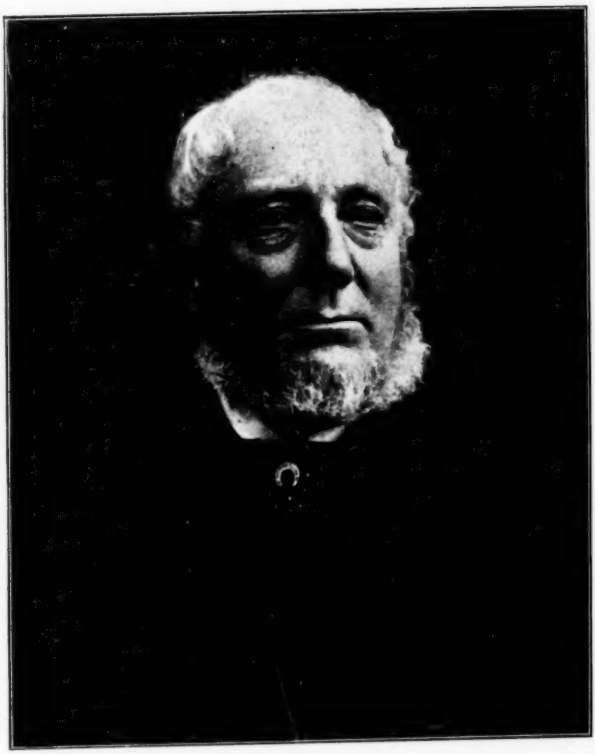

THE LATE MR. HENRY KING SHAW, F.R.C.V.S.